A CLINICAL STUDY ON

"AZHAL KEEL VAYU"

WITH

"PODUTHALAI CHOORANAM"

Dissertation Submitted To

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DEPARTMENT OF POTHU MARUTHUVAM

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Palayamkottai – 627 002.

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Dissertation Topic	A Clinical Study on "**Azhal Keel Vayu Noi"** with evaluation of trial drug "**Poduthalai Chooranam"**
Documents Filed	1) Protocol 2) Data Collection Forms 3)Patient Information Sheet 4) Consent Form
Clinical / Non ClinicalTrial Protocol	Clinical Trial Protocol
Informed Consent Document	Yes
Any other Documents	Case Sheet,Investigation Documents
Date of IEC Approval & its Number	GSMC-II-IEC/2015-Br.-I/07/16.07.2015

We approve the trial to be conducted in its presented form.

The Institutional Ethical Committee expects to be informed about the process report to be submitted to the IEC atleast annually of the study, any changes in the protocol and submission of final report.

16/7/2015

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CERTIFICATE OF BOTANICAL AUTHENTICITY

Certified the following plant drugs used in Siddha formulation PODUTHALAI CHOORANAM (Internal) for the management of "AZHAL KEEL VAYU" *(Osteo arthritis)* taken up for Post Graduation Dissertation Studies by Dr.A. SANTHOSH (Reg.no.321311007) PG Dept, of pothu Maruthuvam are correctly identified and authenticated through Visual inspection / Organoleptic Characters / Experience, Education & Training Morphology Microscopical and Taxonomical methods.

Name	Botanical Name	Family Name	Parts used
Poduthalai	*Phyla nodiflora*	Verbenaceae	Whole plant

Station: Palayamkottai

Date 22/12/15

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CONTENTS

INTRODUCTION

Siddha system of Medicine also known as Siddha Vaidhyam in Tamil Nadu, is the oldest among the Indian Medical Systems namely Siddha, Ayurveda & Unani.

In now a days man is said to be Microcosm and the world, the Macrocosm, because what exists in the world exists in Man. The forces in the Microcosm or Man are identical with forces of the Macrocosm or the world.

This closely follows the Siddhar's doctrine:

"அண்டத்தில் உள்ளதே பிண்டம்"

Kuthambai Siddhar says

"எங்கும் நிறைந்து இருக்கின்ற சோதியை
அங்கத்துள் பார்ப்பாயடி – குதம்பாய்
அங்கத்துள் பார்ப்பாயடி
அண்டத்துக் கப்பால் அகன்ற சுடரினை
பிண்டத்துள் பார்ப்பாயடி – குதம்பாய்
பிண்டத்துள் பார்ப்பாயடி
விண்ணொளியாக விளங்கும் பிரம்மமே
கண்ணொளியானதடி – குதம்பாய்
கண்ணொளியானதடி "

According to Bhoga munivar,

"கோத்துவே எண்சாணந் சடலந்தன்னிற்
கோர்வையாய் நிற்பவார் தானார்தான் கேளு
தேத்துவே அண்டத்திலுள்ள தெல்லாந்
திறமான பிண்டத்திற் காட்டுவேனே."

- போகமுனிவர் ஜெனன சாகரம்

Siddha medicine is the earliest medicine ever documented in the world. Siddha Vaidya can be considered as the crown of all the traditional arts of the ancient world owing to its richness and simplicity.

This system is found and developed by 18 Siddhars. They are the ancient supernatural spiritual saints of India. Among them Sage Agasthiyar is believed to be the foremost of others.

The word Siddha comes from the word "Siddhi' which means an object to attain perfection. Siddha Medicine means medicine that is perfect. Siddha system propounded by the Siddhars is a vast and unique system which defines health as a

perfect state of Physical, Psychological, Social and Spiritual well being of an individual.

Theraiyar describes the characteristics of a Siddha physician as follows:

"அத்த நோக்கினார் அத்த நோக்கினார்
பத்திய முரைப்பார் பத்திய முரைப்பார்
ஆணமுரைப்பார் ஆணமுரைப்பார்
குடிநீருரைப்பார் குடிநீருரைப்பார்
நாடிகைபார்ப்பார் நாடிகைப்பார்ப்பார்
மாத்திரை கடவார் மாத்திரை கடவார்
வாதம் சொல்வார் வாதம் செய்வார்
பித்தமியற்றுவார் பித்தமகற்றுவார்
ஐயமொடுப்பார் ஐயந்தடுப்பார்
மலத்தை தெரிப்பார் மலத்தை பிரிப்பார். "

-தேரன் பாடல் திரட்டு

Siddha Medicine is the only Medical System that is said to bestow immortality. Siddha Medicine revitalizes and rejuvenates the organs and thereby prevents the disease.

A possibility of physical, mental & social well being can be achieved only when the three humours namely Vatha, Pitha, Kabha are maintained in an equilibrium state. Any disturbance of this equilibrium by environmental factors, diet, climatic conditions, our own physical actions can cause an imbalance leading to disease.

This is confirmed by lines of Kuthambai Siddhar

"முக்குற்றம் நீங்க முயலும் மெய்ஞானமே
தக்க மெய்ஞானமடி – குதம்பாய்
தக்க மெய்ஞானமடி "

-குதம்பை சித்தர் பாடல்கள்

Regarding treatmental aspect, Siddha medicine is aimed at keeping the three humours in equilibrium and maintenance of the seven elements namely Saaram, Senneer, Oon, Kozhuppu, Enbu, Moolai, Sukkilam / suronitham.

This is also confirmed by Kuthambai Siddhar poetry .

"முப்பிணிதனை அறியாத மூடர்கள்
எப்பிணி தீர்ப்பாரடி – குதம்பாய்
எப்பிணி தீர்ப்பாரடி
நாடியொருபது நன்காய றிந்திடில்

ஓடிவிடும் பிணியே – குதம்பாய்
ஓடிவிடும் பிணியே
சத்த வகை தாது தன்னையறிந்தோர்
சுத்த வைத்தியனே – குதம்பாய்
சுத்த வைத்தியனே
வாயுவொரு பத்தும் வாய்ந்த நிலைகண்டோர்
ஆயுள் அறிவானடி – குதம்பாய்
ஆயுள் அறிவானடி ”

The drugs are classified under as Thavara (plant products) Thathu (metals, minerals) and sangama (animal products). On the basis of their properties such as suvai (taste), gunam (character), veeriyam (potency), pirivu (class), mahimai (action) the drugs are used. Based upon their mode of utilization the drugs are classified as 32 types of internal medicine & 32 types of external medicine.

The most exclusive part of Siddha medicine is the 8 ways of diagnosis – enn vagai thervugal namely Na, Niram, Mozhi, Vizhi, Sparisam, Malam, Neer, Nadi.

Osteoarthritis is one of the degenerative disease of knee joint. It is more commonly occur in elder people in the age of 4-6 decades. It occurs most commonly in women than in men.

According to our Siddha basis,

“மிகினும் குறையினும் நோய்செய்யும் நூலோர்
வளிமுதலா எண்ணிய மூன்று”……

as said by Sage Thiruvalluvar, the increased or decreased humours can lead to disease. Due to the increased consumption of diet that increases vatha humour and other factors leading to increased vatha humour causes depletion of the enbu and kozhuppu elements. Deranged Vatha humour in knee joints leads to keel vayu. Keel vayu is classified into ten varieties on thridoshic basis. Among them, Azal keel vayu is very common type of keel vayu which we used to come across so many cases in our daily professional life. This thriodoshic damage result in the degenerative disorder – osteoarthritis. The clinical symptoms can similarly correlation in modern medicines is Osteo arthritis Knee Joint. Osteoarthritis is one of the illness due to imbalanced vatha humour. The author here used **Poduthalai chooranam** to overcome the symptoms of knee osteoarthritis. The use of Poduthalai samulam plays an efficient role in cure the Osteoarthritis. According to Gunapadam Mooligai Text Part 1 is clearly mentioned

tha the **Poduthalai Chooranam** is very effective in the management of OA in knee joint.

"பொடுதலையின் பேருரைத்தால் போராமப் போக்கும்
அடுதலைசெய் காசம் அடங்கும் - கடுகிவரு
பேதியோடு சூலைநோய் பேசரிய வெண்மேகம்
வாதமும்போ மெய்யுரக்கும் வாழ்த்து"

- Gunapadam Mooligai – Murugesa Mudaliyar".

(page. no.712) 1st edition

So this drug was selected and further processing was done and given to the patients.

AIM AND OBJECTIVES

The ***"Azhal Keel Vayu"*** is a major ailment of the elderly, nowadays it affects middle aged people also because of their life style and food habits. Though there is no mortality in this disease, its clinical condition worsens in patients and sometimes they become dependant and burden to others which we used to come across so many cases in our daily professional practice.

Keeping all this in author's mind, author tried his best to elucidate a good medicine from ancient Siddha literatures and to create hope and faith in their treatment. This being a preliminary endeavour by the author, it would be a helping hand to the sufferers. With this view this dissertation subject was undertaken.

1. Siddha system of medicine should reach the entire society of the man kind.
2. To expose the unique diagnostic methods mentioned by Siddhars, to know the disease **"Azhal Keel Vayu"** which alters the normal condition under the topic Mukkutram, Poripulangal, Ezhu Udal Kattukkal and Envagai thervugal.
3. To study the clinical case of the disease **"Azhal Keel Vayu"** with keen observation on the Aetiology, Pathology, Diagnosis, Prognosis, Complications and the Treatment by making use of Siddha aspect.
4. To know the role of diet control, medical advices in attaining good results along with the trial medicine.
5. To find the changes in three humours and their thannilai valarchi and Vetrunilai Valarchi in case of **Azhal Keel Vayu**.
6. To know the extent of correlation of Aetiology, Classification, Signs and Symptoms of **Azhal Keel Vayu** in Siddha aspect with Osteo arthritis in Modern medicine.
7. To have an idea about the incidence of the disease with age, sex, socio-economic status and seasonal variations.
8. To have a detailed clinical investigations.
9. To have a clinical trial on **Azhal Keel Vayu** ,with a known specific drug, **Poduthalai Chooranam.**
10. To evaluate the Bio-chemical and Pharmacological effects of trial medicine.
11. To use modern parameters to confirm the diagnosis and prognosis of the disease.
12. To pave way for further research work in future.

ABSTRACT

Since being the commonest disease in the society, number of suffers increasing day by day, So I chosen the disease **"Azhal keel vayu"** for his dissertation work. The evidence of the disease **Azhal keel vayu** is derived from "Sabapathy manuscript of book **Siddha Maruthuvam (pothu)** 6th edition in the year 2004 by K.N.Kuppusamy Mudhaliyar (page no 626)**."**. The signs and symptoms mentioned in "Sabapathy manuscript of Pothu Maruthuvam book**."**. It's closely resembles with that of "**Osteo Arthritis** ' in modern Medicine.

Totally 40 patients was carried out in this study, Among 20 Inpatients and 20 Out patients of both sexes were selected. They were administered with the trial medicine "**Poduthalai Choornam**" 2 gms BD with Hot water after food during the whole study period. Poduthalai Chooranam was choosen for this study with reference from "**Gunapadam Mooligai – Murugesa Mudaliyar**".(page. no.712) 1st edition, by The trial medicine was subjected to biochemical and pharmacological analysis.

At the end of the clinical trial study, the majority of the cases showed good results.

SIDDHA ASPECTS

The concepts of Siddha system are based on fundamental principles of five basic elements of the universe, 96 -Thathuvams, three humours and seven thathus (Physical constituents of the body).

The three humours of the human system are Vatham, Pitham and Kabam .They are called Thrithathu (or) Uyirthathu in their normal condition, which regulate all the physiological activities of the human beings and keep the body healthy.

"வாதமான தேவனே யாதியாகி நின்றவன்
வாதமான தேவனே வையகம மைத்தவன்
வாதமான தேவனே யறுதொழில் வகுத்தவன்
வாதமான தேவனே வண்மைகண்டு கூறுமே"

-சிவவாக்கியர் நாடி.

Among the three humours vadha is the vital humour, other two humours namely, pitha and kapha needs vadha for their role. All movements are due to vadha and that is why it is called Prana of all living being.

When the mutual harmony of the 3 humours is disturbed they are called thridosha and they bring about ill health.

Thiruvalluvar says,

"மிகினும் குறையினும் நோய் செய்யும் நூலோர்
வளிமுதலா எண்ணிய மூன்று"

- திருவள்ளுவர்

Based on this theory, the problems of various systems are classified as Vatha diseases, Pitha diseases and Kaba diseases.

கீல் வாயு

சித்த மருத்துவத்தில் எண்பது வகை வளி நோய்கள் பற்றி கூறப்பட்டுள்ளது. அழல் கீல் வாயு என்பது, கீல்வாயு என்ற தலைப்பின் கீழ் கூறப்பட்ட பத்து வகைகளுள் ஒன்று. கீல்வாயு என்பது மூட்டு மற்றும் அதனைச் சுற்றியுள்ள பகுதிகளில் ஏற்படும் நோய்களை குறிப்பதாகும்.

According to **Sabapathy manuscript,** Azhal keelvayu comes under the classification of ten Keelvayus. In Keelvayu mostly the deranged factor is Vatham. So Keelvayu comes under the vatha diseases according to thridosha theory. It is also confirmed by

Agasthiar in Agasthiar Gunavagadam.

"தானாக கீல்வாத ரோகம் பேரை

.....

நோய் தனக்கு பாகியாய் வாதரோக மென்பார்

நுட்பமுள்ள வாதரோக மெண்பதுந் தான்

ஆய்ந்தெடுத்து இதற்குள்ளே அடக்கம் பாரு

....."

-அகத்தியர் குணவாகடம்

வேறு பெயர்கள் :-

சந்துவலி, மூட்டுவலி, மேகசூலை, முடக்கு வாயு, ஆமவாதம், சந்து வாதம், சூலைகட்டு, சந்திக சிலேஷ்ம ரோகம், வாதசூலை, வாயுரோகம்.

கீல் வாயு

"தானான கீல்வாத ரோகம் பேரை

சாற்றுகிறேன் நீயறிய விபர மாக

மானான வாய்வுரோகம் வாத ரோகம்

மகத்தான முடக்குவாய்வு முடக்கு வாதம்

தேனான சந்தீக சிலேட்டும ரோகம்

தெளிவான கைகாலில் பிடிப்பு ரோகம்

ஊனான ரசவாதம் சூலைக் கட்டு

உத்தமனே சந்திவாதம் வாதசூலை யாமே

ஆமென்ற இத்தனையும் அதற்குப் பேராம்"

.....

-அகத்தியர் குணவாகடம்

காரண பெயர்கள் :-

நோய் காரணம்	:	மேக சூலை
முக்குற்ற நிலை மாறுபாடு	:	வாத சூலை
		சந்திக சிலேஷ்ம ரோகம்
இடத்தை கொண்டு	:	சந்து வாதம்
		மூட்டு வலி
		சந்து வலி
		ஆம வாதம்
குறிகுணங்களை கொண்டு	:	சூலைகட்டு
		முடக்குவாதம்.

Description of the nomenclature

Azhal keel vaayu = Azhal + Keel + Vaayu

Azhal = Pitham

Keel = Joint

Vaayu = Vatham

Initially the joint is affected by the vitiated vatham. Pitham and kabam accompany later. It is a disease which is common in pitha kaalam (middle 1/3 of the lifespan).

The principle causative factor for all the joint diseases (Keelvayu) is the Vatha dosha and this is quoted by various siddhars as follows:

‘‘காணப்பா வாதமீறில் கால்கைகள் பொருத்து நோவும்

பூணப்பா குடல்புரட்டும் மலசலம் பொருமிக்கட்டும்’’

-அகத்தியர் வைத்திய காவியம் 1500 ,பாடல் எண் - 10 ,பக்கம் எண் 2

‘‘தக்கவாயு கோபித்தால் சந்துவுளைந்து தலைநோவா

மிக்கமூரி கொட்டாவி விட்டங்கெரியு மலங்கட்டும்

ஒக்கநரம்பு தான்முடங்கு முலர்ந்து வாய்நீரூறிவரும்’’

-தேரையர் வாகடம், பாடல் எண் - 42 ,பக்கம் எண் - 13

‘‘வாதவீறு அன்னமிறங்காது கடுப்புண்டாம் வண்ணமுண்டாம்

மோதுகட்டுரோகம் சுரமுண்டா மிருமலுமா முறங்காதென்றும்

ஓதுசூரிய வாதமனலாகு நடுக்கமுண்டாம் பொருள்களாய்ந்

தீதெனவே நரம்பிசித்து சந்துகள்தோறும் கடுக்குந்தினமுந்தானே’’

-தேரையர் வாகடம் ,பாடல் எண் 210 ,பக்கம் எண் 58

‘‘எறிய நல்வாதம் எறிக்கும் குணங்கேளு

குறியெனக் கைகால் குளைச்சு விலாச் சந்து

புறியென நொந்துடல் பச்சைப்புண் ஆகுமே:

-திருமூலர் கருக்கிடை வைத்தியம் - 600 ,பாடல் எண்-36, பக்கம் எண் -11

நோய் இயல்

கீல்வாயு என்னும் வளி நோயானது வலி, வீக்கம், குத்தல், மூட்டுகளை அசைக்க சிரமம், விறைப்புத்தன்மை, சுரம், பசியின்மை, சோகை ஆகிய குறிகுணங்களை உடையதாகும்.

“வளியுமையந் தன்னிலை கெட்டு
வலியுடன் வீக்கச் சுரமும் காய்ந்து
மூட்டுகள் தோறும் முடுக்கியே நொந்து
மூட்டுகள் தன்னில் நீரும் சுரந்து
தாங்கொணா வலியுமா நொந்திடுமம்மே”

-சபாபதி கையேடு. சித்த மருத்துவம் (பொது)
,பக்கம் எண் 624

வாதமும், கபமும் தன்னிலைக் கெடுவதால் மூட்டுகளில் வலி வீக்கம், நீர்கோர்த்தல் ஆகியவை பிறக்கின்றன.

Clinical Features are:

1. Painful knee joints
2. Swelling
3. Restricted knee joint movements
4. Stiffness
5. Fever
6. Loss of appetite
7. Synovial effusion

நோய் வகைகள்

1. வளி கீல் வாயு
2. **அழல் கீல் வாயு**
3. ஐய கீல் வாயு
4. வளி அழல் கீல் வாயு
5. அழல் வளி கீல் வாயு
6. வளி ஐய கீல் வாயு
7. அழல் ஐய கீல் வாயு
8. ஐய வளி கீல் வாயு
9. ஐய அழல் கீல் வாயு
10. முக்குற்ற கீல் வாயு

Noi Varum Vazhi (Aetiology)

According to Siddha system ,causes of diseases are due to the disturbance of thrithathus. In Keelvayu the chief deranged factor among the thrithathu is the **Vatham.** The derangement of vatham occurs under various conditions. They are

- Environmental factors
- Physical factors
- Factors of kanmam

a) Environmental factors

"ஆடியாதியாய் ஐப்பசி ஈறாய்
அனிலமதற் கோரரசியல் காலம்"

-சதக நாடி (நோய் நாடல் பாகம் I ,பக்கம் எண் - 167,168)

Sathaganaadi describes that the vatha diseases are predominant in the months of Aadi to Iypasi.

"வாதவர்த்தனை காலமேதோ வென்னில்
மருவுகின்ற ஆனி கற்கடகமாகும்
ஆதவைப் பசியோடு கார்த்திகை தன்னில்
அடருமே ....."

-யூகி சிந்தாமணி, பாடல் எண் 245, பக்கம் எண் 76.

The vatha dosha provokes in its own site in the Tamil months Aani and Aadi (தன்னிலை வளர்ச்சி). But it provokes and spread beyond its site in the month of Iypasi and Karthigai (வேற்றுநிலை வளர்ச்சி) and reassumes its normalcy in the rest of the year (தன்னிலை அடைதல்).

According to another concept,

"பதுமத்தைப் பூக்க வைக்கும் பானுமிக்க காயும்
முதுவேனி லிற்பு விநீர் முற்றும் - கதுமென
வற்றும் கப‡கும் வாயுமிகும்....""

- மருத்துவர் தனிப்பாடல்

முதுவேனிற் காலத்தில், சூரிய வெப்பத்தின் காரணமாக பெரும் வாரியாக நீர் ஆவியாக்கப்பட்டு பூமியில் வறட்சி நிலவும். அதுபோல் நமது உடலில் வறட்சி ஏற்பட்டு வளிநோய் வருவதற்கு ஏதுவாகிறது.

b) Physical factors:

1. ‘‘வளிதரு காய்கிழங்கு வரைவிலா தயிலல் கோழை
முளிதரு போன்மிகுக்கு முறையிலா உண்டி கோடல்
குளிர்தரு வளியிற் தேகங்குனிப்புற வுலவல் பெண்டிர்
களிதரு மயக்கம் பெற்றோர் கடிசெயல் கருவியாமால்’’

-சபாபதி கையேடு – சித்த மருத்துவம் (பொது) ,பக்கம் எண் 624

Excessive intake of certain roots and vegetables that produce vatha diseases, improper food intake, prolonged exposure to cold air, staying in hill stations, over indulgence in sexual activity and hereditary factors produce Keelvayu.

2. ‘‘தானான கீல்வாத ரோகம் பேரை
……
போமே தான்ரச தூஷியத் தினாலே
பொல்லாத இந்த நோய் காணும் பாரு’’

-அகத்தியர் குணவாகடம்

According to **Agasthiyar gunavagadam,** keelvayu occurs due to dietary substances which degrade the quality of chyle (அன்னரசம்).

3. **‘‘**பகரவே வாதமது கோபித்தப்போ
பண்பாக பெண்போகம் அதுதான் செய்யில்
நகரவே வெகுதூர வழிநடக்கில்
நளிரான காற்றுமே பனிமேல் பட்டால்
மிகரவே காய்கள் கனிகிழங்கு தன்னை
மிகவருந்தி மீறியே தயிர்தான் கொண்டால்
முகரவே முதுகெலும்பை முறுக்கி நொந்து
முழங்காலும் கணுக்காலும் கடுப்புண்டாமே’’

-யூகி சிந்தாமணி, பாடல் எண் 215, பக்கம் எண் 89

Indulging in the sexual act during vitiation of vatha, walking for a long distance, prolonged exposure to dampness and cold, harmful consumption like taking excessive curd after eating fruits, vegetables and tubers produces toxic factors which affects bone and muscles and produce vatha diseases

4. ‘‘தானென்ற கசப்போடு துவர்ப்பு கைப்பு
சாதகமாய் மிஞ்சுகினும் சமைத்த வன்னம்
ஆனென்ற ஆறினது புசித்த லாலும்
ஆகாயத் தேறலது குடித்தலாலும்
பானென்ற பகலுறக்க மிராவிழிப்பு

பட்டினியே மிகவுறுதல் பாரமெய்தல்
தேனென்ற மொழியார் மேற்சிந்தை யாதல்
சீக்கிரமாய் வாதமது செனிக்குந்தானே''

-யூகி சிந்தாமணி, பாடல் எண் 244 ,பக்கம் எண் 76

Excessive intake of bitter, astringent and salty food ,drinking rain water, irregular sleep pattern , undue starving, strain due to excessive weight lifting and sexual perversion induce vatha diseases.

5. ''வெய்யிலில் நடக்கையாலும் மிகத்தண்ணீர் குடிக்கையாலும்
செய்யிழை மகளிரைச் சேர்ந்தன பவிக்கையாலும்
பையனே உண்மையாலும் பாகற்காய் தின்கையாலும்
தையலே வாதரோகம் சனிக்கு மென்றறிந்து கொள்ளே''

- தேரையர் வாகடம், பாடல் எண் 16, பக்கம் எண் 5

Excessive walking in hot sun, excessive intake of water, excessive indulgence in sexual activity, intake of bitter guard etc., may disturb the normal functions of vatham

6. ''தொழில்பெறு கைப்புக் கார்த்தல் துவர்த்தல் விஞ்சுகினுஞ் சோறும்
பழையதாம் வரகு மற்றைப் பைந்திணை யருந்தி னாலும்
எழில்பெறப் பகலு றங்கி இரவினி லுறங்கா தாலும்
மழைநிகர் குழலி னாளே வாதங்கோ பிக்குங் காணே''

-பரராச சேகரம்

Improper dietary habits &sleep pattern causes vatha diseases.

7. "காலங்கண் மாறி யுண்ணுங் காரியத் தாலுந் தண்ணீர்
சாலவே யருந்தி னாலுஞ் சந்தியி லுட்கார்ந் தாலும்
வாலவார் முலைநல் லாளே வாதமுற் பவிக்குங் காயே"

-பரராச சேகரம்

Factors of Kanmam :

In Siddha system, many diseases are said to be precipitated by kanma, which means the deeds good or bad committed, by an individual in his previous and the present births. According to ***Agasthiyar kanma kandam – 300*** Vatha diseases may also be precipitated by kanmam.

Vatha Kanma varalaru says,

"நூலென்ற வாதம் வந்த வகைதானேது
துண்மையாய்க் கன்மத்தின் வகையைக் கேளு
காலிலே தோன்றியது கடுப்பதேது
கைகாலில் முழக்கியது வீக்கமேது
கோலிலே படுகின்ற விருட்சமான
குழந்தை மரந்தனை வெட்டல் மேல்தோல்சீவல்
நூலிலே சீவஜந்து கால் முறித்தல்
நல்லகொம்பு தழைமுறித்தல் நலித்தல் தானே"

-அகத்தியர் கன்ம காண்டம் ,பாடல் எண் 56, பக்கம் எண் 23

Psychological factors such as removing the bark of living trees, injuring the animals, cutting the branches in the living trees and plucking the leaves may produce vatha diseases.

Due to Karmic Law:

"அந்தணர் கற்பு மாதர் அருளிய சாபத்தாலும்
முந்திய வினையாலும் முதிர்கர்ப்ப மேகத்தாலும்
சிந்தையிற் கொடுமையாலும் சிவகுரு நிந்தையாலுந்
தொந்தமாம் வியாதியாலும் தோன்றிடும் சூலைதானே"

-அகத்தியர்

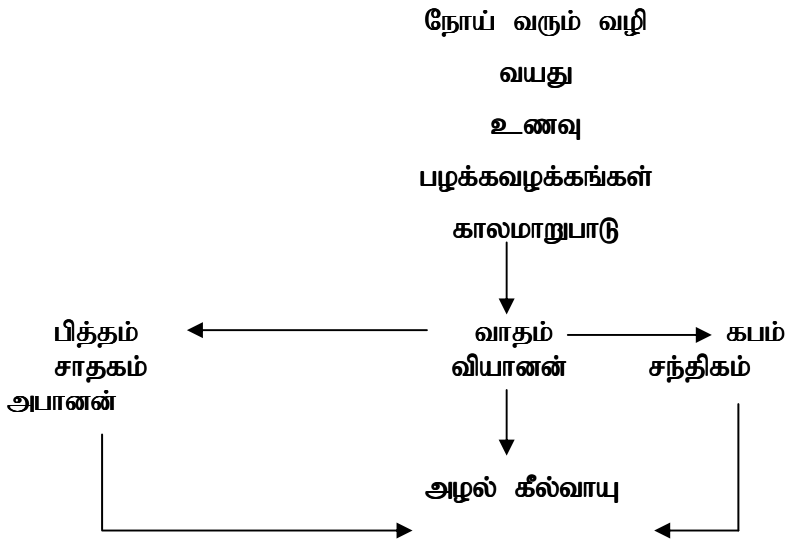
Soolai (Neuritic pain) may also occur by the curse of well-characterized people and ladies or due to evil deeds in the previous births or due to megam produced by their parents or due to bad thoughts and curse of his Guru.

Prodromal symptoms of Keelvayu:

Nasal block, running nose, hoarseness of voice, low grade fever, Painful arthralgia are the prodromal symptoms of Keelvayu.

Individual derangements in Mukkutram ie Vatham, Pitham and Kabam:

- Abanan and viyanan are affected in vatham.
- In pitham, Saathaga pitham is affected.
- Santhigam is affected in Iyyam.



வாதம் மிகு குணம் :

"அறியவிம் மூன்றின் தன்மை சொன்னார்நந்தி
எறிய நல்வாத மெறிக்குங் குணங்கேளு
குறியெனக் கைகால் குவைச்சு விலாச்சந்து....."

-திருமூலர் கருக்கிடை வைத்தியம் 600

"வாதவீறு அன்னமிறங்காது கடுப்புண்டாம் வண்ணமுண்டாம்
மோதுகட்கு ரோகம் சுரமுண்டா மிருமலுமா முறங்காதென்றும்
ஓதுதரிய வாதமனலாகு நடுக்கமுண்டாம் பொருள் களயர்ந்த
தீதெனவே நரம்பித்து சந்துகள் தோறுங்கடுக்குந் தினமுந்தானே"

- தேரையர் வாகடம் ,பாடல் எண் 210, பக்கம் எண் 58

"தக்க வாயு கோபித்தால் சந்துளைந்து சூலைநோவா
மிக்க கொட்டாவி விட்டங் கெரியு மலங்கெட்டும்
ஒக்க நரம்பு தான் முடங்கு மலர்ந்து வாய் நீரூறிவரும்
மிக்க குளிரும் நடுக்கமாய் மேனி குன்றி வருங்கானே"

- தேரையர் வாகடம் ,பாடல் எண் 42, பக்கம் எண் 13

வாதம் மிகும்போது பசியின்மை, உடல் கடுப்பு, சுரம், இருமல், உறக்கமின்மை, உடல் நடுக்கம், நரம்புத்தளர்ச்சி, சந்துகள் தோறும் குடைதல் விலாச் சந்துகள் நோதல், வயிறு பொருமல், குடலிறைச்சல், மலச்சிக்கல் மிகுந்த கொட்டாவி, போன்ற குறிகுணங்கள் தோன்றும்.

When the Vatha kutram aggravates it will produce the following signs and symptoms:

- loss of appetite
- excruciating pain

- fever
- cough
- Insomnia
- nervous weakness
- joint pain *(*Myalgia)
- Intercostal Neuralgia
- Dyspepsia
- Flatulence
- Chills or Rigor
- Constipation

AZHAL KEELVAYU

Azhal Keelvayu is one among the 10 types of Keelvayus.

When the vatha dosha is in vitiated condition, activity and foodstuffs, which provoke the pitha dosha is the causative factor of the Azhal keelvayu.

The normal structural qualities (வடிவத்தன்மை) of the Pitha are

- Heat (வெப்பம்)
- Sharpness (கூர்மை)
- Lubrication (நெய்ப்பு)
- Relaxation (நெகிழ்ச்சி)
- Motion (இயக்கம்)

Azhal keelvayu in deranged pitha dosha may produce joint stiffness, restriction of movement and crepitation in the affected joints.

"தானான கீல்வாத ரோகம் பேரை
……..
பொல்லாத இந்த நோய் காணும் பாரு
நாமேதான் முழங்கால்கள் பெரியகீல்கள்
நன்மையுடன் அதைச்சுற்றி இருக்கும் சவ்வின்
……."

-அகத்தியர் குணவாகடம்

According to ***Agasthiyar gunavagadam,*** keelvayu affects knee joints and other major joints and their periarticular surface.

In Azhal keelvayu the Muzhangal poruthu (knee joint) is commonly affected and produce disability. So i select ***Azhal keelvayu in Muzhangal poruthu for an elaborate study*** as my dissertation work.

Clinical features of Azhal keelvayu:

According to Sabapathy manuscript ,

"பித்தக்கீல் வாய்வு தன்னாற் பிறங்குகீன் முட்டு வீங்கிச்
சித்தர் மருத்து வத்துஞ் சீர்படாத் தன்மைத் தாகித்
தத்தறு காய்ச்சல் கண்டு சாலவே தனைதான் தந்தே
மெத்தறு சிகிச்சை தன்னால் மென்மெல நீங்கு மப்பா"

-சபாபதி கையேடு - சித்த மருத்துவம் (பொது), பக்கம் எண் - 626

- முட்டிகளில் வீக்கம்
- தீக்குற்ற மிகுதியால் கீல்களின் பசை வறண்டு, பசையற்று கீல் அசையும் போதெல்லாம் ஒலி உண்டாகும்.

- சில வேளைகளில் கீல்களிலுள்ள பொருத்துகள் ஒன்றொடொன்று ஒட்டிக்கொண்டு மடக்க முடியாமலே நின்றுவிடும்.

Review of literature about the diseases of Knee joint implies that the clinical features more or less correlates with Azhal keelvayu.

According to Theriyar,

"கிடைவாதம் புடைவாதம் பிறளுவாதம் கேழ்க்கி
லேகமன வாதமெண்பத் தொன்று -***பாடல் எண் 209 ,பக்கம் எண் 57***

.....

தானாகுங் சந்துவாதம் சதியின் வாதம் தான்
கெண்டைவாத முழங்கால்வாதமாமே

–பாடல் எண் 205, பக்கம்எண் 56

.....

முழங்கால் வாதங்கனைத்து வீக்கமாகி முசியாமல்
கடுத்து நடக்கொட்டா தென்று "

- பாடல் எண் 217, பக்கம் எண் 60
-தேரையர் வாகடம்

According to **Theriyar vagadam** ***vatha*** diseases are 81 types. Muzhangal vatham is one of the vatha diseases. It is characterized by pain and swelling in the knee joint and inability to walk.

According to Sekaraja Sekaram ,

"வருந்தவேமுழங்கால்வீங்கி வலித்துநொந்துளைந்துகுத்திப்
பொருந்தவேமுடக்கிமிண்டிப் போதவேநடைகொடாது
திருந்துவேல்கணைமானம்பு சேலெனமிளிருங்கண்ணாய்
இருந்தயர்முழங்கால்வாதக் குணமிதென்றியம்பினாரே"

-செகராச சேகர வைத்தியம், **பாடல் எண் 1 ,பக்கம்எண் 106**

In Segaraja sekara vaidhyam among the 85- vatha diseases, Muzhangal vatham is characterized by pain and swelling in the knee joint, restricted movement and difficulty in walking.

Diagnosis in Siddha:

Piniyari muraigal (Method of Diagnosis) is based upon three main principles namely

- Poriyal Arithal
- Pulanal Arithal
- Vinaathal

Poriyal Arithal (Inspection):

"Poriyal arithal" means examining the patient by the "Pori" of the physician for proper diagnosis. Pori is considered as the

"Five sensory organs" of perception namely,

- Mei (Skin)
- Vai (Tongue)
- Kan (Eye)
- Mookku (Nose)
- Sevi (Ear)

ஞானேந்திரியங்களின் ஆய்வு :

செவி	ஒலியை அறிய செய்தல்	இயல்பு
மெய்	உடலில் ஊற்றை அறிதல்	முழங்கால் மூட்டுகளில் வீக்கம், வலி
கண்	ஒளியை அறிய செய்தல்	இயல்பு
நாக்கு	சுவையை அறிய செய்தல்	இயல்பு
மூக்கு	வாசனை நுகர செய்தல்	இயல்பு

கண்மேந்திரியங்களின் ஆய்வு :-

வாய்	பேச செய்யும்	இயல்பு
கை	இடுதலும், ஏற்றலும் செய்யும்	இயல்பு
கால்	நடக்கச் செய்யும்	முழங்கால் மூட்டுகளில் வலி, நடக்க சிரமம்
எருவாய்	மலத்தை கழிக்கும்	மலச்சிக்கல்
கருவாய்	கரு, சுக்கிலத்தைக் கழிக்கும்	இயல்பு

Pulanal arithal (Palpation):

Pulan are five senses they are,

- Smell
- Taste
- Vision
- Sensation of touch
- Hearing

"Pulanal arithal" means examining the "Pulan" of the patient by the Physician to diagnose a disease.

Vinaathal (Interrogation):

Vinaathal is questioning for gathering information regarding the history of disease and its clinical features which are very essential to diagnose the disease.

ENVAGAI THERVUGAL (Eight diagnostic Tools):

It is a unique method of diagnosis in Siddha system of medicine. They are clearly explained by Siddhar Theraiyar

"நாடி ஸ்பரிசம் நா நிறம் மொழி விழி
மலம் மூத்திரம் மருத்துவராயுதம்"

- நோய் நாடல் நோய் முதல் நாடல் (முதல் பாகம்) ,பக்கம்எண் 270

1.Naadi (Pulse):

Among the envagai thervugal naadi is the most important one. Naadi is felt as vatham, pitham and kabam with the tip of the index ,middle and ring fingers respectively over the end of the radius.

Normally vatham, pitham and kabam are held in the ratio of 1: ½:¼. Derangement in this will reflect as disease.

Naadi nadai in keelvayu:

''திருத்தமாம் வாதத் தோடே தீங்கொடு பித்தஞ் சேரில்
பொருத்து கள்தோறும் நொந்து போதவே பிடிக்கும் சூலை''

-நோயின் சாரம் - சித்த மருத்துவம் (பொது) ,பக்கம்எண் 634

''காணப்பா வாத மீறில்
கால்கைகள் பொருத்து நோகும்''

- காவிய நாடி - சித்த மருத்துவம் (பொது) ,பக்கம்எண் 634

''சொல்லிய வையத்தோடு பித்தமுங் கூடிற்றானால்
வல்லியம் போலக் குத்தும் மைந்தனே எலும்பு தோறும்''

- காவிய நாடி - சித்த மருத்துவம் (பொது), பக்கம்எண் 634

''அறிந்துபார் வாதமே தனித்தானதால்
சரிந்திடவே கால் முடக்கும்''

- அகத்தியர் ரத்தினச் சுருக்கம் ,பக்கம் எண் 634.

''வாதத்தில் சேத்துமமாகில் வலியோடு வீக்கமுண்டாம்''

- அகத்தியர் நாடி

In Azhal keelvayu the following naadinadai are commonly felt.

- ❖ Vatham
- ❖ Vathapitham
- ❖ Pithavatham

2. **Sparisam (Sensation to touch):**

In Azhal keel vaayu mild heat noticed over the affected joint.

3. Naa (Tongue):

In Azhal keel vaayu no abnormality is seen in Naa.

4. Niram(Colour):

In Azhal keel vaayu, some skin colour changes seen in affected area due to inflammatory mechanism.

5. Mozhi (Voice):

In Azhal keel vaayu no abnormality is seen.

6. Vizhi (Eyes):

In Azhal keel vaayu no abnormality is seen.

7. Malam (Faeces):

In Azhal keel vaayu constipation was reported in some cases.

8. Moothiram (Urine):

In urine, Neerkkuri and Neikkuri examinations are done.

Neikkuri:

Prior to the day of urine examination the patient is instructed to take a balanced diet and quantities of food must be proportionate to his appetite. The patient should have no disturbed sleep. After waking up in the morning, the first urine voided is collected in a clear wide mouthed glass dish or China clay bowl and is subjected to analyse of "Neerkkuri and Neikkuri" within one and a half an hour of its collection.

The collected urine specimen is kept in a glass dish or china clay container and observed under direct sunlight without shaking the vessel. Then add one drop of gingely oil and observe the spreading pattern and conclude as follows,

"அரவென நீண்டின .ஃதே வாதம்
ஆழிபோற் பரவின் அ.ஃதே பித்தம்
முத்தொத்து நிற்கின் மொழிவ தென் கபமே"

- நோய் நாடல் பாகம் I, பக்கம் எண் 298, 299

b. Neerkkuri:

"வந்த நீர்க்கரி யெடை மணம் நுரை எஞ்சலென்

றைந்திய லுளவை யறைகுது முறையே"

- சித்த மருத்துவாங்க சுருக்கம் ,பக்கம் எண் 510

In Urine examination the following characteristic features are observed namely

- Niram - Colour
- Edai - Specific Gravity
- Manam - Smell
- Nurai - Frothy nature
- Enjal - Quantity of urine voided

Apart from these, frequency of micturition, abnormal constituents, such as sugar, protein, blood stains, pus, crystals also to be found out.

In Azhal keel vaayu straw or hay coloured urine was noticed in Neerkkuri.

PARUVAKAALAM (Seasonal variations):

S.No	STATE OF KUTTRAM	KAALAM
1.	Vatham thannilai adaithal	Munpani kaalam, pinpani kaalam,koothir kaalam, elavenil kaalam
2.	Vatham thannilai valarchi	Muthuvenil kaalam
3.	Vatham vetrunilai valarchi	Karkaalam

முதுவேனிற் காலத்தில் நமது உடலில் வறட்சி ஏற்பட்டு வளிநோய் வருவதற்கு ஏதுவாகிறது.

திணை (Geographical distribution)

குறிஞ்சி : மலையும் மலை சார்ந்த பகுதியும்

முல்லை : காடும் காடு சார்ந்த பகுதியும்.

மருதம் : வயலும் வயல் சார்ந்த பகுதியும்

நெய்தல் : கடலும் கடல் சார்ந்த பகுதியும்

பாலை : மணலும் மணல் சார்ந்த பகுதியும்.

முல்லை மற்றும் நெய்தல் நிலங்களில் வாத நோய்கள் பெருமளவில் ஏற்படும்.

ஏழு உடல் தாதுக்களின் ஆய்வு :

SL. No	UDAL KATTUKAL	INCREASED CONDITIONS	DECREASED CONDITIONS
1.	Saaram	Loss of appetite, excessive salivation	Tiredness, Fatigue Diminished activity of the sense organs.
2.	Senneer	Boils and tumours in different Parts of the body, Spleenomegaly,colic pain,increased blood pressure reddish eye and skin. Jaundice,Leprosy,Haematuria etc	Tiredness, Lassitude, Anaemia
3.	Oon	Tumours or extra growth around the neck, face, abdomen, thigh, Genitalia etc., with dyspnoea	Muscle wasting
4.	Kozhuppu	Tumours or extra growth around the neck, face, abdomen,thigh, Genitalia etc., with dyspnoea and loss of activity	Pain
5.	Enbu	Extra growth of bones and teeth	Weak bones, teeth, nails and hair.
6.	Moolai	Heaviness, swollen eyes, Swollen phalanges, oliguria and non healing ulcers	Osteoporotic changes
7.	Sukkilam or Suronitham	Increased sexual activity and Symptoms as that of urinary calculi	Infertility, pain in genitalia

In Azhal keel vaayu,

Saaram, Kozhuppu, Moolai and Enbu thathukkal are commonly affected.

Saaram : Weakness, pain in knee joints

Kozhuppu : Morning stiffness occurs in affected knee joints

Enbu : Pain occur in affected knee joints, crepitations Present.

Moolai : Osteophytic changes, extra osteophytic formation are seen in the few joints

MUKKUTRAM:

Uyir thathukkal ie Vatham, Pitham and kabam are responsible for normal physiological conditions of the body. Vatham is mainly responsible for proper loco-motor functions. Bones and joints are considered to be the main site of vatha.

In Azhal keel vaayu the vatha kutram is mainly affected followed by pitham and kabam. This produces the following signs and symptoms,

- Deranged viyanan leads to painful stimuli and difficulty in movements.
- Deranged Abanan leads to constipation.
- Inflammatory changes of the joints, heat, redness and swelling are developed due to altered pitham.
- Sathaga pitham gets affected hindering the loco motor functions.
- Along with vatham, kabam is also deranged,Santhikam is affected and this leads to abnormality in joint movements.
- Sclerosing of bone margin, increased secretion of synovial fluid may lead to synovial effusion due to increased kabam.

NAME	LOCATION	PHYSIOLOGICAL FUNCTIONS
Abanan	Lower abdomen and Extremities	Responsible for urination, defaecation and parturition, Menstruation, ejaculation of the sperm.
Viyanan	Heart	Responsible for movements of all parts of the body and sensation.
Samanan	Stomach	Responsible for proper digestion

NOI KANIPPU VIVATHAM (DIFFERENTIAL DIAGNOSIS):

Azhal keel vaayu is differentiated from the following diseases,

1. VALI KEEL VAAYU:

"வலிக்குத்தல் வீக்கங்காணும் வாய்த்தொண்டை வறட்சி காய்ச்சல்
தலைவலி மார்துடிப்புத் தாங்கொணா வலி வீக்கந்தான்
நிலவு காங்கணுக் குறங்கு நீடு தோள் முழங்கைக் காற்காம்
மலக் குடற்கட்டு வேர்வை வாதக்கீல் வாயு விதாமே"

➢ ***சபாபதி கையேடு சித்த மருத்துவம் (பொது), பக்கம் எண் 625***

The clinical features of valikeel vaayu are intolerble pain and swelling involving major joints and associated systemic disturbances like dryness of mouth, pyrexia, headache, palpitation, constipation and sweating. In advanced cases it may affect the heart and produce "Thamaraga vaayu" .In modern science it is compared to Rheumatic carditis .

2. IYA KEEL VAAYU:

"கருதருங் கபக்கில் வாயு கண்டிடின் உடலிளைக்கும்
உருமெலிவாக்குங் கொள்ளும் உண்டியைச் சுருக்கு மினபந்
தருதுயில் நீங்கு முட்டிற் றாங்கொணா வலுவையாக்கும்
இருமலே விக்கல் வாந்தி, சோபை பாண்டெழுப்பும் பாரே"

➢ ***சபாபதி கையேடு சித்த மருத்துவம் (பொது) ,பக்கம் எண் 627***

It is characterized by severe pain in the joints associated with loss of weight, anorexia, insomnia, cough, hiccough, vomiting, anaemia and dropsy. The common sites are spinal cord, hip joints and knee joints .In modern science it is compared to tubercular arthritis.

3. VALI IYA KEEL VAAYU:

"அவையம் வாதக் கபக்கீல் வாயுவான் வலி மிகுந்தே
உயங்கு நீர் கோத்து கீல்கள் ஓரியின் தலைபோற் காணும்
நயங்கொள்ள முடக்கல் நீட்டல் நண்ணிடாமெய்யுங்காயும்,
மயக்குறு முறக்மின்னாம் மன்னிய நெரிக்கட்டாமே"

- சபாபதி கையேடு சித்த மருத்துவம் (பொது), பக்கம் எண் 628

It is characterized by pain in the joints associated with effusion of joint fluid and swelling, restricted joint movements, pyrexia, fainting, insomnia, especially in knee joint asymmetrically, lymphadenopathy, generalized malaise, atrophy of the affected limb etc. The affected joint looks like "Fox's Head". In modern science it is compared to Charcot's joint .

LINE OF TREATMENT

In Siddha system the main aim of the treatment is to cure Udarpini (due to Mukkuttram) and Manapini (due to changes in Mukkunam). Treatment is not only for perfect healing but also for the rejuvenation and prevention of the diseases.

Line of management is as follows:

- Kaapu (Prevention)
- Neekkam (Treatment)
- Niraivu (Restoration)

KAPPU (Prevention):

"புளிதுவர் விஞ்சுங்கறி யாற்பூரிக் கும்வாதம்
ஒளியுவர் கைப்பேறில் பித்துச் சீறும்"

- ***நோய்நாடல் பாகம் I, பாடல் எண் 2 ,பக்கம் எண் - 23***

The preventive methods for Azal keel vaayu are as follows:

1. Reduce the excess body weight by diet and physical exercise.
2. Modify the nature of work which gives stress to a particular joint. e.g. - Avoid prolonged standing and walking.
3. Avoid the sedentary life style
4. Avoid excessive intake of sour, astringent and bitter tasted foods.

NEEKKAM (Treatment in Siddha):

Neekkam:

"மூன்றிலொன் றுயர்ந்ததை முன்னரறிந்து
முந்தியதனை யொழித்திடு மருந்திடு
தணியும் நோயின் தந்திரமிதுவே
பேணிக்கணித்திடின் பிறவாய் பின்குணம்"

- ***நோய்நாடல் பாகம் I, பக்கம் எண் 139***

The aim of Neekkam is based on

"விரேசனத்தால் வாதம் தாழும்"

"வமனத்தால் பித்தம் தாழும் "

"நசிய அஞ்சனத்தால் கபம் தாழும்"

To bring the deranged humours to normal equilibrium state.

To treat the patient with internal medicine and external medicine.

1. PURGATIVE:

In Azhal keel vaayu vatha kutram is deranged. So a purgative vellai ennai – 15ml with warm water is given in early morning in empty stomach on the first day of treatment .

2. INTERNAL MEDICINE:

Poduthalai Chooranam – 2 gm twice a day with hot water after food

3. DIETARY RESTRICTIONS:

இச்சா பத்தியத்தில் நீக்கும் பொருள்கள்:

"கடுகு நற்றிலத் தெண்ணெய் கூழ்ப் பாண்டங்கள் கடலை
வடுவதாகிய தெங்குமா வருக்கை நற்காயம்
மடிவி வாத வெள்ளுள்ளி கொள் புகையிலை மதுபெண்
இடறு பாகலோ டகத்தி நீக்கிடலிச் சாபத்தியம்"

- ***சித்த மருத்துவாங்க சுருக்கம்.***

கடுகு, எள்நெய், கலியாண பூசணிக்காய், கள், கடலை, தேங்காய், மாங்காய், பலா, காயம், பூண்டு, கொள், புகையிலை, பெண்கள் சேர்க்கை, பாகல், அகத்தி இவைகளை இச்சா பத்தியத்தில் நீக்க வேண்டும்.

"புளி துவர் விஞ்சும் கறியால் பூரிக்கும் வாதம்"

ஆதலால் புளிப்பு, துவர்ப்பு சுவையுள்ள உணவு வகைகளை நீக்க வேண்டும்.

சேர்க்கத்தக்கன :

"செய்கழு நீர்கோடைந் தேன் மிளகு நல்லெண்ணெய்
தங்கு பெருங்காயந் தழுதாழை - எங்கெங்கும்
கூட்டு சிறுமுத்துநெய் கோதில் உளுந்திவைகள்
வாட்டு மனி லத்தை மதி"

- ***பதார்த்த குண சிந்தாமணி***

செங்கழுநீர், கோட்டம், குறிஞ்சிதேன், மிளகு, பெருங்காயம், தழுதாழையிலை, சிற்றாமணக்கு நெய், உளுந்து கத்திரி பிஞ்சு, முருங்கைப் பிஞ்சு, அவரைப்பிஞ்சு, வாழைப் பிஞ்சு, முளைக்கீரை, நெய், பால், மோர் இவற்றை சேர்க்கலாம். **முளைகட்டிய பயிறு வகைகள் சிறந்தது.**

நோயின்றி வாழ வழிமுறைகள் :-

நாள் ஒழுக்கம்:

1. மலச்சிக்கல், வயிறு பொருமல் இன்றி பார்த்துக் கொள்ள வேண்டும்.
2. காய்கறி, பழங்கள், முளைகட்டிய தானியங்கள் ஆகியவற்றை அதிகமாக உணவில் சேர்த்து கொள்ள வேண்டும்.
3. சுண்ணசத்து நிறைந்த உணவு பொருள்களை சேர்த்துக் கொள்ள வேண்டும்.
4. பகல் உறக்கம் கொள்ளக் கூடாது.
5. உடற்பயிற்சிகளை செய்து உடல் எடையை சீராக்க வேண்டும்.
6. காய்ந்து ஆறின வெந்நீர், நீர் கலந்த புளிப்பில்லா மோர் போன்றவை நாளொன்றுக்கு 3 லிட்டர் குறையாமல் அருந்த வேண்டும்.

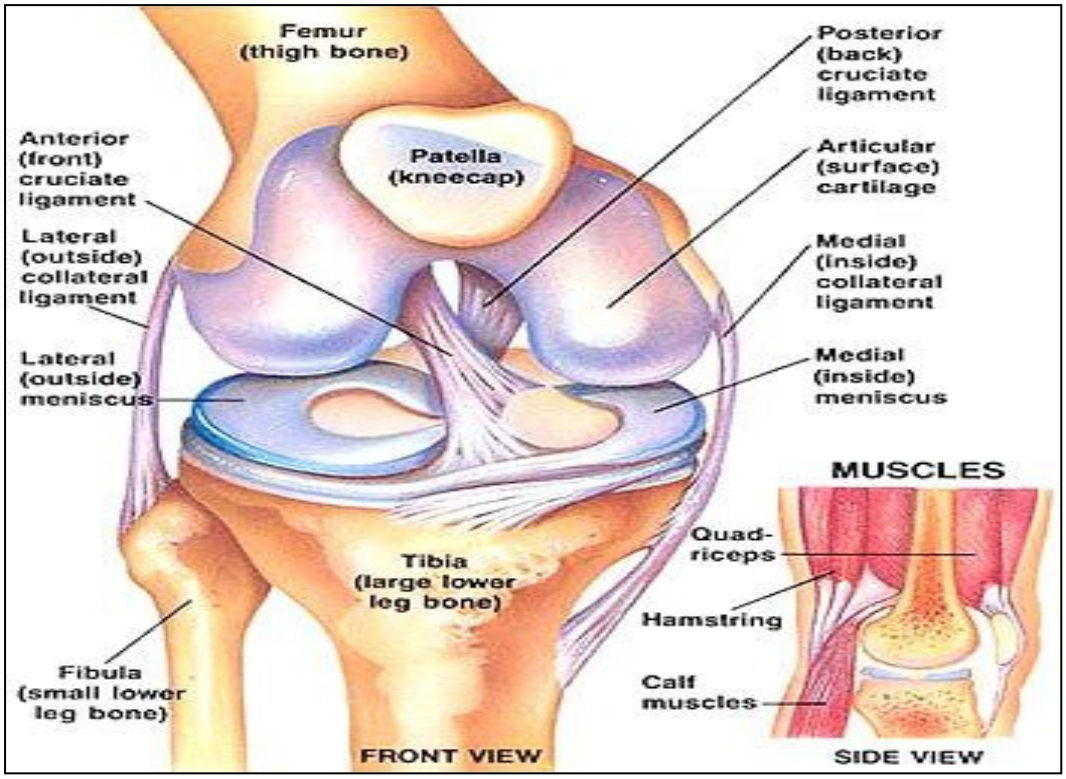
கால ஒழுக்கம்:

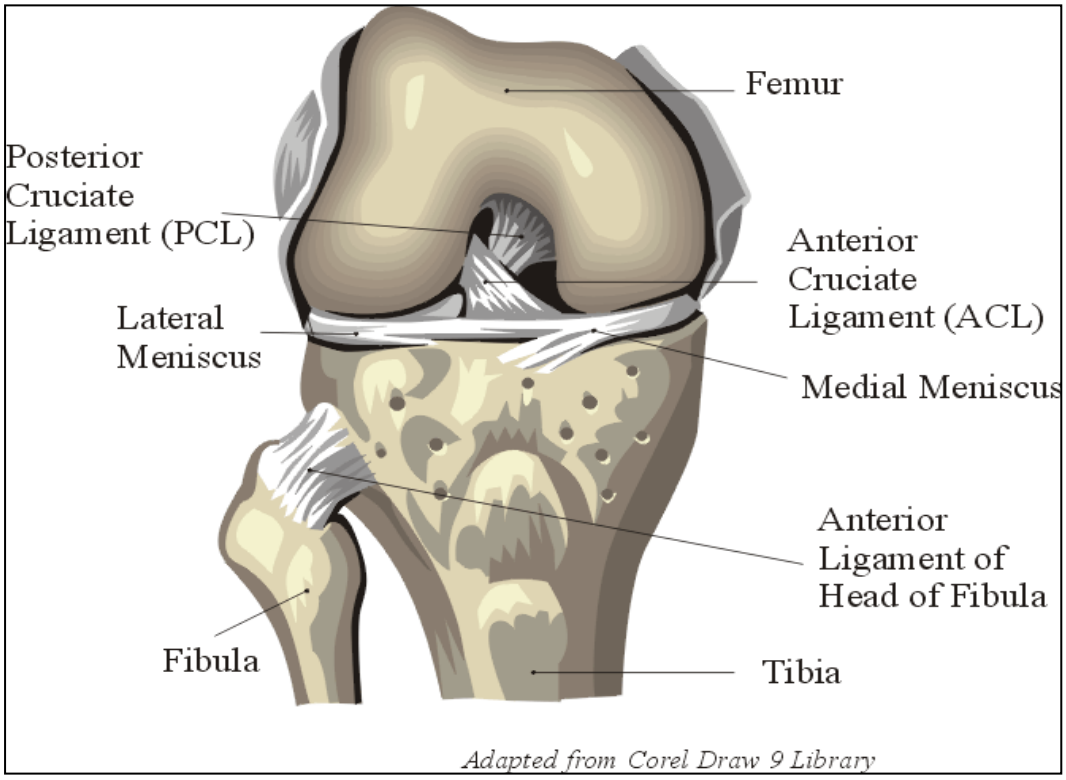
1. குளிர்காற்றில் ஈடுபடக் கூடாது.
2. வாரம் ஒரு முறை எண்ணெய் குளியல் செய்ய வேண்டும்
3. 45 நாட்களுக்கு ஒரு முறை நசியம் செய்து கொள்ள வேண்டும்
4. நான்கு மாதங்களுக்கு ஒரு முறை பேதி மருந்து உட்கொள்ள வேண்டும்.
5. 6 மாதங்களுக்கு ஒரு முறை வமன மருந்து உட்கொள்ள வேண்டும்.

Niraivu:

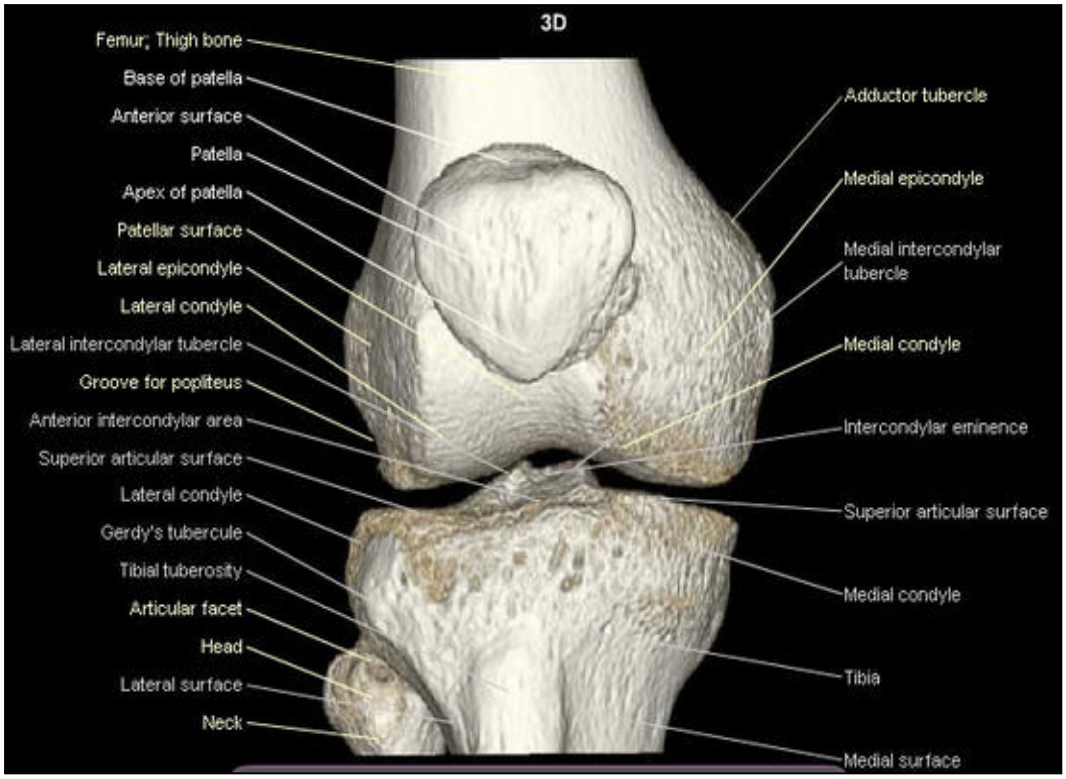
Reassurance of disease recovery were given to all the patients. All the patients were advised to live in a good moral and peaceful life.

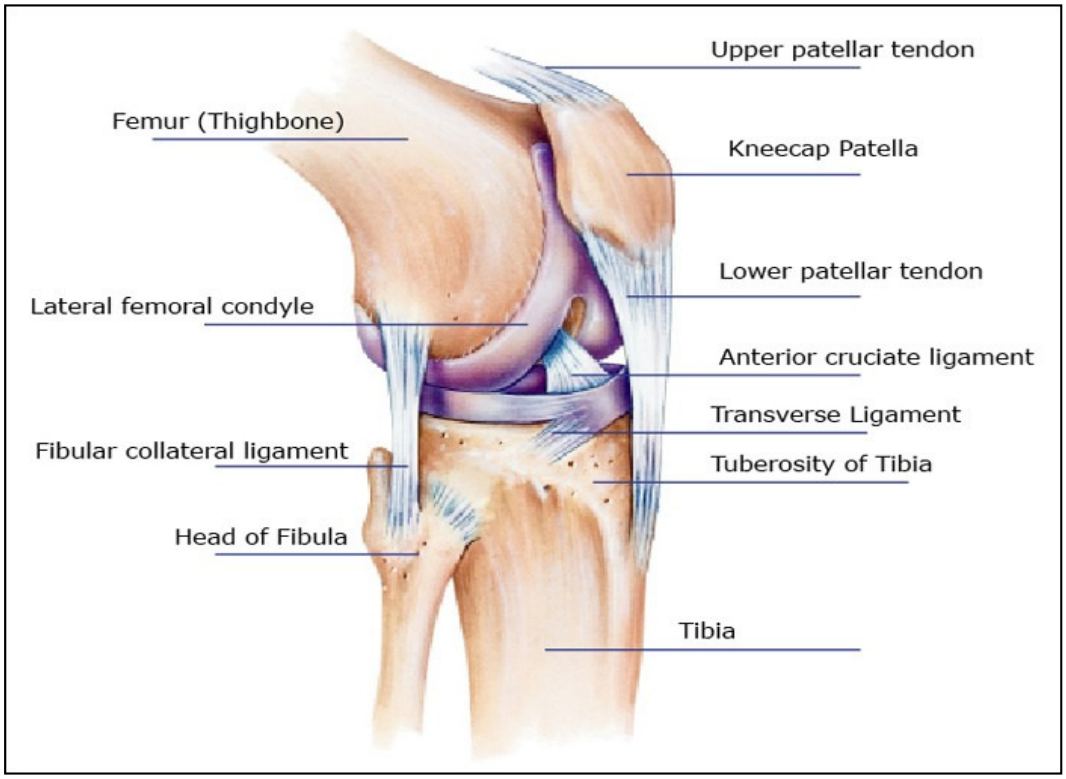
ANATOMY OF KNEE JOINT





ANATOMY OF KNEE JOINT





MODERN ASPECT

Anatomy of the knee joint

Knee is the largest and most complex joint of the body. The complexity is the result of fusion of three joints namely the lateral femorotibial,medialfemorotibial and femoropatellar joint.

Type:

It is a compound synovial joint, incorporating two-condylar joint between the condyles of femur and tibia and one saddle joint between femur and patella.

Knee joint is a hinge joint formed by the condyles of the femur, the condyles of the tibia and the posterior surface of the patella. The anterior part of the capsule consists of the tendon of the quadriceps femoris muscle which also supports the patella.

Articular surfaces:

Knee joint is formed by

- Condyles of femur
- Condyles of tibia
- The patella

The femoral condyles articulate with tibial condyles below and behind, and with the patella in front. Structurally, knee is a weak joint because the articular surfaces are not congruent. The tibial condyles are too small and shallow to hold the large, convex femoral condyles. The femoropatellar articulation is also quite insecure because of their shallow surfaces, and also the outward angulation between the axis of thigh and leg. The stability of the joint is maintained by various ligaments.

Blood supply:

Knee joint is supplied by

- Genicular branches of popliteal artery
- Descending genicular branch of femoral artery
- Descending branch of lateral circumflex femoral artery
- Two recurrent branches of anterior tibial artery.
- Circumflex fibular branch of posterior tibial artery.
- Descending branch of lateral circumflex femoral artery
- Two recurrent branches of anterior tibial artery.
- Circumflex fibular branch of posterior tibial artery.

Nerve supply:

1. Femoral nerve, through its branches to vasti.
2. Sciatic nerve, through the genicular branches of tibial and common peroneal nerve.
3. Obturator nerve, through its posterior division

Movements of knee joint

Active movements at the knee are

- Flexion
- Extension
- Medial rotation
- Lateral Rotation .

Flexion and extension are the chief movements of much greater range than rotations. These are permitted in the upper compartment of the joint, above the menisci. Flexion and extension take place in transverse axis. During extension, the axis moves upwards and forwards. During flexion, the axis moves downwards and backwards.

Rotatory movements at the knee are of a smaller range than that of flexion and extension. Rotations take place around a vertical axis and are permitted in the lower compartment of the joint, below the menisci.

Integrity of the knee joint:

Lateral motion and rotatory motion of the knee joint in extension is controlled by the capsule, collateral ligaments and cruciate ligaments, in flexion, by the same structures except the fibular collateral ligament.

Forward gliding of the tibia on the femur is controlled by the anterior cruciate ligament and the quadriceps.

Backward gliding of the tibia on the femur is controlled by the posterior cruciate ligament and the posterior capsule.

Lateral gliding of the tibia on the femur is controlled by tibia, inter condylar spine and the femoral condyles with the aid of all the ligaments.

Hyper extension is controlled by both the collateral ligaments, both cruciate ligaments, both menisci, the posterior aspect of the articular capsule, the oblique popliteal ligament and the architecture of the femoral condyles.

Hyperflexion is controlled by both cruciate ligaments, both menisci, the femoral attachment of the posterior aspect of the capsule, the femoral attachment of both heads of gastrocnemius muscle and the bony structure of the condyles of the femur and the tibia.

The menisci cushion both hyper extension and hyper flexion. The tibial collateral ligaments are closely related to the medial meniscus but there is no strong fibrous tissue attachment between them. The tibial collateral ligament glides forward and backward in extension and flexion.

Articular Cartilage:

The ends of the bones in a synovial joint are covered with a layer of articular cartilage. This is an avascular tissue that consists of cartilage cells (chondrocytes) embedded in a thick matrix of proteoglycans, water, type II collagen and smaller amount of other proteins.

Although there is no cell division in normal cartilage, chondrocytes are metabolically active cells that are responsible for synthesis and turnover of cartilage matrix throughout life. The matrix consists of a meshwork of type II collagen fibrils that run through a hydrated gel of proteoglycan molecules, the most important of which is aggrecan.

Aggrecan consists of a core protein, to which several glycosaminoglycan side chains are attached.The most important glycosaminoglycans, are chondroitin sulphate and keratan sulphate. Cartilage also contains hyaluronan, a long glycosaminoglycan which consists of multiple glucuronic acid and N-acetyl galactosamine disaccharide repeats.

The expansive force of the negatively charged and hydrated aggrecan combined with the restrictive force of the collagen meshwork, gives articular cartilage excellent shock- absorbing properties.

With ageing, the amount of chondroitin sulphate decreases whereas that of keratin sulphate increases. The end result is reduction in water content and impairment of cartilage's shock-absorbing properties. Age related changes in cartilage differ from those found in osteoarthritis, where there is abnormal chondrocyte division, loss of proteoglycan from matrix and an increase in water content.

Cartilage matrix is constantly being turned over and in health a perfect balance is maintained between synthesis and degradation of matrix components.

Zones of articular cartilage:

1. Superficial layer (tangential zone)
2. Superificial zone is the first to show changes of osteoarthritis.
3. Transitional layer
4. Deep radial layer
5. Calcified cartilage layer

Muscles producing movements

Movement	Principal muscles	Accessory muscles
A. Flexion	1. Biceps femoris 2. Semitendinosus 3 .Semimembranosus	l.Gracilis 2.Sartorius 3.Popliteus 4.Gastrocnemius
B. Extension	Quadriceps femoris	Tensor fascia lata
C. Medial rotation of flexed leg	1.Popliteus 2.Semimembranosus 3.Semitendinosus	l.Sartorius 2.Gracilis
D. Lateral rotation of flexed leg	Biceps femoris	

OSTEOARTHRITIS:

Osteoarthritis, also referred to as degenerative joint disease, degenerative arthritis, or hypertrophic arthritis, is among the most frequent and symptomatic medical problems for middle aged and older people. It affects people of all ethnic groups in all geographic locations, occurs in both men and women but commonly in women, and it is the most common cause of long term disability in patient populations older than 65 years. More than one third of people older than 45 years report joint symptoms that vary from a sensation of occasional joint stiffness and intermittent aching associated with activity to permanent loss of motion and constant deep pain. Those who are most severely affected have crippling joint deformity and

instability. The joint degeneration that causes the clinical syndrome of osteoarthritis occurs most frequently in the hand, foot, knee, hip, and spine joints, but it can develop in any synovial joint.

Though osteoarthritis affects many joints, knee joint is the most common which we come across in our day today practice.

Synonyms:

Osteo arthrosis, Degenerative joint diseases. Hypertrophic arthritis.

Definition:

Osteoarthritis is defined as a condition of synovial joints characterized.

By,

- ❖ Focal loss of articular hyaline cartilage.
- ❖ Simultaneous proliferation of new bone with remodeling of joint contour.

The rate of degeneration is greater than the rate of repair of articular cartilage. Osteoarthritis, however, is not a disease or a single condition. It is best viewed as the dynamic repair process of synovial joints, some but not all of which result in symptomatic **'Joint failure'.** As there is no evidence of synovial thickening or inflammatory infiltration, in uncomplicated conditions, the term **'Osteoarthrosis'** is preferred to, Osteoarthritis by Kellgren (1961)

Epidemiology:

Osteoarthritis is the most common joint disease of humans. Osteoarthritis preferentially targets only certain small and large joints, the knee and hip are the principal sites of significant disability. Knee osteoarthritis is more prevalent than hip osteoarthritis. Under the age of 55 years the joint distribution of osteoarthritis in men and women is similar. In older individuals,hip osteoarthritis is more common in men. Knee osteoarthritis is more common in women than in men. Osteoarthritis of inter- phalangeal joints and the thumb base is more common in women.

Age is the most powerful risk factor for osteoarthritis. In radiographic survey of women less than 45 years old only 2% had osteoarthritis; between the ages of 45 to 64 years, however, the prevalence was 30% and for those older than *65* years it was 68%. In males the figures were similar but somewhat lower in the older age groups.

Risk factors for osteoarthritis

- ❖ Female sex
- ❖ Age
- ❖ Genetic factors
- ❖ Major joint trauma
- ❖ Repetitive stress (eg) vocational
- ❖ Obesity
- ❖ Congenital or developmental defects
- ❖ Prior inflammatory joint disease
- ❖ Metabolic or endocrine disorders.

Age:

With increasing age, the cells synthesize smaller aggrecans and less functional link proteins, leading to the formation of smaller irregular proteoglycan aggregates.

Their mitotic synthetic activities decline with age, and they become less responsive to anabolic cytokines and mechanical stimuli.

Excessive Repetitive Joint Loading:

Maintenace of normal synovial joint structure and function requires regular joint use. Life long normal daily activities and regular recreational running have not been shown to increase the risk of joint degeneration. There is evidence, however, that reptitive loading of normal joints can exceed the tolerance of a joint and cause degeneration.

Occupation

Farmer	:	Hip OA
Miner	:	Knee and spine OA
Cotton wool workers	:	Hand OA.
Jack hammer operator	:	Elbow OA
Vibrating tool Worker	:	Upper limb OA

Sports activities :-

Athlets	:	HipOA, Knee OA.
Foot baller	:	Knee OA
Boxers	:	Hand OA may be due to trauma
Soccer Playing	:	Knee and hip OA.

Nutritional:

Vitamin C is important in the development of normal cartilage. Deficiency of vitamin C might lead to the development of weak cartilage.

Vitamin D is also important for bone. Deficiency increases the risk of joint space narrowing and progression of the disease .

People with low mineral density such as in osteoporosis may be at increase risk of osteoarthritis.Adequate intake of **calcium** as recommended for age and gender can help to maintain bone density.

Sex Hormones:

Osteoarthritis occurs more frequently in women after the age of 50 than in men of same age. Epidemiologic studies of women who take oestrogen replacement therapy report that these woman are less likely to have osteoarthritis than women not taking oestrogen.

Obesity:

Obesity increases the risk of developing osteoarthritis. Obese weight people might reduce their chances for developing or aggravating osteoarthritis by losing weight. Further more if person already has substantial osteoarthritis in a weight bearing joint such as knee or hip, weight reduction can significantly improve their ability to rehabilitate after joint surgery as well as reduce their risk of surgical complications.

- Rheumatoid arthritis and septic arthritis may progress to osteoarthritis
- **Prolonged NSAID**

Aetiological classification of Osteoarthritis

I. **Idiopathic**

A. Localized Osteoarthritis.

1. **Hands:** Herbeden's and Bouchard's nodes (nodal), erosive inter phalangeal arthritis (non nodal), 1st carpometacarpal joint.
2. **Feet:** hallux valgus, hallux rigidus,contracted toes, talonavicular.
3. **Knee:**
    a. Medial compartment.
    b. Lateral compartment.
    c. Patellofemoral compartment

Hip:

a. Eccentric (superior).
b. Concentric (axial, medial)
c. Diffuse (coxae senilis)

Spine:

a. Apophyseal joints.
b. Intervertebral joints (discs)
c. Spondylosis
d. Ligamentous (Hyperostosis, Forestier's disease)

Other single sites:

(eg): glenohumeral, acromioclavicular, tibiotalar, sacroiliac, temporomandibular.

B. Generalized Osteoarthritis

It includes 3 or more of the areas listed above

II. Secondary:

A. Trauma:

- Acute
- Chronic (occupational, sports)

B. Congenital or developmental:

1. **Localized diseases:** congenital hip dislocation, slipped epiphysis, Perthes' disease.
2. **Mechanical factors:** unequal lower extremity length, valgus/varus deformity.
3. **Bone dysplasias:** epiphyseal dysplasia, spondyloepiphyseal dysplasia.

C. Metabolic:

- Ochronosis (alkaptonuria)
- Hemochromatosis
- Wilson's disease
- Gaucher's disease

Endocrine:

- ❖ Acromegaly
- ❖ Hyperparathyroidism
- ❖ Diabetes mellitus
- ❖ Obesity
- ❖ Hypothyroidism.

E. Calcium deposition diseases:

- ➢ Calcium pyrophosphate dihydrate deposition.
- ➢ Apartite arthropathy.

F. Other bone and joint diseases:

1. Localized: fracture, avascular necrosis, infection, gout.

2. **Diffuse:** Rheumatoid arthritis, Paget's disease, osteopetrosis, osteochondritis.

G. Neuropathic (charcot's joints)

H. Endemic:

1. Kashinbeck
2. Mseleni

I. Miscellaneous:

1. Frostbite
2. Caisson's disease
3. Hemoglobinopathies

Causes of young onset Osteoarthritis (<45 years)

Monoarticular

- ➢ Previous trauma.
- ➢ Localized instability.

Pauciarticular or polyarticular.

- ➢ Prior joint disease (juvenile idiopathic arthritis)
- ➢ Metabolic or endocrine diseases (Hemochromatosis, ochronosis,Acromegaly).
- ➢ Epiphyseal dysplasia.
- ➢ Late avascular necrosis

- Neuropathic joint.
- Endemic Osteoarthritis.

Monoarticular and pauciarticular Osteoarthritis:

It is the'classic' form of Osteoarthritis which presents with pain and dysfuncion in one or two of the large weight bearing joints. There may be an obvious underlying abnormality:- acetabular dysplasia, old perthes' disease or slipped epiphysis, a previous fracture or damage to ligaments or menisci.

Polyarticular (generalizd) Osteoarthritis:

It is far and away the most common form of Osteoarthritis, though most of the patients never consult an orthopaedic physician. The patient is usually a middle-aged woman who presents with pain, swelling and stiffness of the finger joints. The first carpo metacarpal and the big toe metatarsophalangeal joints, or the knees and lumbar facet joints, maybe affected as well.

The changes are most obvious in the hands. The inter-phalangeal joints become swollen and tender, and in the early stages they often appear to be inflamed. Over a period of years, osteophytes and soft tissue swelling produce a characteristic knobby appearance of the distal interphalangeal joints (Heberden's nodes) and less often the proximal interphalangeal joints (Bouchard's nodes); pain may disappear but stiffness and deformity can be disturbing. Some patients present with painful knees or backache and the knobby fingers are noticed only in passing. There is a strong association with carpal tunnel syndrome and isolated tenovaginties.X-rays show the characteristic features of Osteoarthritis usually maximal in the distal interphalangeal joints of the fingers.

Known causes of Joint Degeneration (Secondary Osteoarthritis)

Cause	Presumed Mechanism
Intra –articular fractures	Damage to articular cartilage and/or joint incongruity
High –intensity impact joint loading	Damage to articular cartilage and/or subchondral bone
Ligament	Joint instability

Joint dysplasias (developmental and hereditary joint and cartilage dysplasias)	Abnormal joint shape and/or abnormal articular cartilage
Aseptic necrosis	Bone necrosis leads to collapse of the articular surface and joint incongruity
Acromegaly	Overgrowth of articular cartilage produces joint incongruity
Paget's disease	Distortion or incongruity of joints caused by bone remodeling, joint instability
Ehlers – Danlos syndrome	Joint disability
Gaucher's disease (hereditary deficiency of the enzyme, glucocerebrosidase, leading to accumulation of glucocerebroside)	Bone necrosis or pathologic bone fracture leading to joint incongruity
Sticker's syndrome (progressive hereditary arthro-opthalmopathy)	Abnormal joint and /or articular cartilage development
Joint infection (inflammation)	Destruction of articular cartilage
Hemophilia	Multiple joint hemorrhages
Hemochromatosis (excess iron deposition in multiple tissues)	Mechanism unknown
Ochronosis (hereditary deficiency of the enzyme homogentisic acid oxidase, leading to accumulation of homogentisic acid)	Deposition of homogenitisic acid polymers in articular cartilage
Calcium pyrophosphate deposition disease	Accumulation of calcium pyrophosphate crystals in articular cartilage
Neuropathic arthropathy (Charcot's joints, syphilis, diabetes mellitus, syringomyelia, meningomyelocele, leprosy, congenital insenstivity to pain, amyloidosis)	Accumulation of calcium pyrophosphate crystals in articular cartilage. Loss of proprioception and joint sensation results in increased impact loading and torsion, joint instability and intra-articular fractures.

Generalized Osteoarthritis:

Generalized osteoarthritis is characterized by involvement of three or more joints or groups of joints (distal interphalangeal and proximal interphalangealjoints are counted as one group each). Herbeden's and Bouchard'snodes are prominent. Symptoms may be episodic, with "flare-ups" of inflammation marked by soft tissue swelling, redness, and warmth.

Marked female preponderance. Peak onset in middle age

Erosive Osteoarthritis:

Distal and /or proximal interphalangeal joints of the hands are most predominently affected. Erosive osteoarthritis tends to be more destructive than typical nodal osteoarthritis.

Osteoarthritis in unusual sites

Osteoarthritis is uncommon in the shoulder, elbow, wrist, metacarphophalangeal joints, ankle and the lesser toes.

Endemic Osteoarthritis:

Osteoarthritis occasionally occurs as an endemic disorder affecting entire communities. It may be due to either some environmental factors peculiar to that region or to an underlying generalized dysplasia in a genetically isolated community.

Kashin-Beck disease:

It is seen in the northern parts of Russia and China. It usually manifests as a generalized Osteoarthritis of the finger joints, elbows, knees and ankles. Shortness of stature is common suggesting that the condition starts in childhood. It remains uncertain whether the widespread bone deformity and cartilage degeneration are due to a genetic disorder, an unusual dietary deficiency or the ingestion of mycotoxins in spoiled wheat.

Mseleni joint disease:

It is found among the Tsonga people along the eastern seaboard of southern Africa. Osteoarthritis of the hips is common but numerous other joints are affected as well, leading to crippling deformities in older adults.

Rapidly destructive osteoarthritis:

Every so often a patient with apparently straightforward osteoarthritis shows rapid and startling progression of bone destrucion, quite out of keeping with the usual slow progress of osteoarthritis. Usually it is the hip that is affected, but similar changes may be seen in other joints. Names such as analgesic arthropathy and indomethacin hip reflect the belief that is due to the pain-dampening effect of strong anti-inflammatory drugs. These drugs also suppress the prostaglandin synthesis and they might theoretically, inhibit healing of microfractures in osteoarthritic joints.

Alternative explanations are that the condition is a form of crystal arthropathy-crystal deposition being quite common in osteoarthritis or that it is due to osteoporosis and bone necrosis supervening on pre-existing osteoarthritis. The fact that most of the patients are elderly women favours the latter hypothesis.

Neuropathic joint disease (Charcot's disease):

The most destructive arthropathy is that associated with lack of pain sensibility and position sense. Neuropathic joints lack the normal reflex safeguard against abnormal stress or injury and the subchondral bone disintegrates with progressive deformity. The joint is neither warm nor particularly tender, but swelling is marked, fluid is greatly increased and bits of bone may be felt everywhere. The forces are greatly redistributed by angular deformity & Obesity.Obesity plays a larger role in the aetiology of the most serious cases of knee osteoarthritis.

Pathology:

Pathogenesis and Pathology:

Osteoarthritis usually involves all of the tissues that form the synovial joint, including articular cartilage, sub- chondral and metaphyseal bone, synovium, ligaments, joint capsules, and the muscles that act across the joint; however, the primary changes consist of loss of articular cartilage, remodeling of subchondral bone, and formation of osteophytes. The earliest articular cartilage structural changes seen in osteoarthritis are fraying or fibrillation of the superficial zone extending into the transitional zone and violation of the tidemark by blood vessels from subchondral bone.

The cardinal features are

- Progressive cartilage destruction
- Subarticular cyst formation
- Sclerosis of the surrounding bone
- Osteophyte formation
- Capsular fibrosis

Frequently the condition is initiated as chondro malacia patellae (chondromalacia-cartilage softening). Due to continuous friction, the joint surface of patello femoral joint is eroded and degenerated.

Degeneration of hyaline cartilage is the primary lesion. The cartilage is progressively eroded and bone matrix is exposed. The erosion is patchy with normal islands of cartilage in between. The cartilage and perichondrium around the periphery of joint are stimulated which leads to elevation of nonarticular surface of joint above the remaining surface and later on projects circumferentially to give **'lipping'** appearance. There is synovitis with fibrosis and sub synovial connective tissue.

According to Harrison, there is proliferation of blood vessels, which leads to increased blood supply to subchondral bone with thinning of overlying cartilage due to pressure.

Cartilage is degenerated, which later on invaded by large blood vessels and finally replaced by bone. Edema in subchondral marrow is followed by formation of mucinous fatty marrow and dilation of surrounding sinusoids. In center of these area mucoid secretion occurs. These cyst cavities expand by resorption of bone trabeculae. Osteoblastic activity surrounds these areas, which forms the sclerotic wall. According to other theories, herniation of synovial fluid through cracks within denuded subchondral bone leads to cyst formation.

Outward cartilage growth, followed by ossification and local periosteal new bone formation mainly around the capsular attachments, leads to **"osteophytic lipping".**

Inflammation and metaplasia of synovial membrane occurs later on. Detached flakes of cartilage and metaplastic synovium give rise to cartilaginous and osteocartilaginous **"loose bodies".**

Menisci are also degenerated which are extremely vulnerable to injury there after. Minimal or gross tears may occur. Though, in cruciate ligaments degeneration takes place, generally they remain intact even in severe osteoarthritis. There is hypertrophy of infrapatellar pad.

The capsule and synovium are often thickened but cellular activity is slight, however, sometimes there is marked inflammation or fibrosis of the capsular tissues.

Pathogenesis:

Articular cartilage is the major target tissue in osteoarthritis. Articular cartilage serves two essential functions within the joint.

They are

- ❖ It provides a remarkably smooth bearing surface. So that, with joint movement, the bones glide effortlessly over each other.
- ❖ It prevents the concentration of stress, so that the bones do not shatter when the joint is loaded.

Cartilage Changes:

Many of the mechanisms responsible for progressive loss of cartilage in degenerative joint disease remain unknown, but the process can be divided into three over lapping stages as described below.

Stages in the development and Progression of Osteoarthritis

Stage I	**Cartilage Matrix Disruption or Alteration**
	Disruption or alteration of the matrix macromolecular framework associated with an increase in water concentration may be caused by mechanical insults, degradation of matrix macromolecules, or alterations of chondrocyte metabolism. Initially, the type II collagen concentration remains unchanged, but the collagen meshwork may be damaged, and the concentration of aggrecan and the degree of proteoglycan aggregation decrease.
Stage II	**Chondrocyte Response to Matrix Disruption or Alteration**
	When chondrocytes detect a disruption or alteration of their matrix they can respond by increasing matrix synthesis and degradation and by proliferating. Their response may restore the tissue, maintain the tissue in an altered state, or increase cartilage volume. They may sustain an increased level of activity for years.

sStage III	Decline In the Chondrocyte Response
	Failure of the chondrocyte response to restore or maintain the tissue leads to loss of articular cartilage accompanied or preceded by a decline in the chondrocytic response. The causes for the decline in chondrocytic response remain poorly understood, but it may be partially the result of mechanical damage to the tissue, with injury to chondrocytes and a down regulation of the chondrocyte response to anabolic cytokines.

In the first stage, either before or with the appearance of fibrillation, the matrix macromolecular framework is disrupted or altered at the molecular level and the water content increases. Early in this process, the chondrocytes increase their expression of enzymes that can degrade the matrix macromolecules. Although the concentration of type II collagen remains constant, decrease in proteoglycan aggregation and concentration and in the size of the glycosaminoglycan chains usually accompany the increase in water content. These changes increase the permeability (the ease with which water and other molecules move through the matrix) and decrease the stiffness of the matrix, alterations that may increase the vulnerability of the tissue to further damage.

The **second stage** begins when chondrocytes detect the tissue damage such as degradation of matrix macromolecules or alterations in osmolarity, charge density, or strain and release mediators that stimulate a cellular response that is often quite brisk. The response consists of both anabolic and catabolic activity as well as chondrocyte proliferation. Anabolic and mitogenic growth factors presumably have an important role in stimulating synthesis of matrix macromolecules and chondrocyte proliferation clusters or clones of proliferating cells surrounded by newly synthesized matrix molecules constitute one of the histologic hallmarks of the chondrocytic response to cartilage degeneration. Inflammatory cytokines such as interleukin (IL)-1 and tumor necrosis factor alpha (TNF-α) probably also influence chondrocyte activity because chondrocytes produce these molecules in response to a variety of mechanical and chemical stress. IL-1 induces formation of nitric oxide, which diffuses rapidly and stimulates the activity of matrix metalloproteinases (MMPs), enzymes that degrade matrix macromolecules. Degradation of type IX and type XI collagens and other molecules may destabilize the type II collagen fibril meshwork, leaving many of the

type II fibrils intact initially, but allowing expansion of aggrecan and increased water content. Disruption of the superficial zone, a decline in aggregation, and an associated loss of aggrecan because of enzymatic degradation increase the stress on the remaining collagen fibril network and chondrocytes with joint loading. Enzymatic degradation also clears damaged and intact matrix components and may release anabolic cytokines previously trapped in the matrix that stimulate synthesis of matrix macromolecules and chondrocyte proliferation.

During the second-stage of osteoarthritis, enzymes that degrade articular cartilage matrix molecules have a critical role in causing progressive joint degeneration. MMPs destroy aggrecan and activate collagenase, which destroys collagen. **The balance between degradative enzyme activity and matrix synthesis determines the rate of articular cartilage loss**.

In this second stage of osteoarthritis, which may last for many years, the repair response exceeds the synthesis.

Failure to stabilize or restore the articular cartilage leads to the third stage in the development of osteoarthritis, progressive loss of articular cartilage, and a decline in the chondrocytic anabolic and proliferative response. This decline could result from mechanical damage and death of chondrocytes no longer stabilized and protected by a functional matrix, but it also appears to be related to, or initiated by, a down regulation of chondrocyte response to anabolic cytokines.

Bone Changes:

- Alteration of the subchondral bone
- Degeneration of articular cartilage includes increased subchondral bone density
- Formation of cyst-like bone cavities
- Appearance of regenerating cartilage within and on the subchondral bone surface
- Osteophyte formation

Clinical features:

Pain:

Initially pain increased by exertion and relieved by rest

Later pain in bed at night, less in the morning and worse in the evening after days activity.

Pain may increase with changes in the weather especially storms or a drop in temperature.

Causes of Pain:

Since articular cartilage is aneural, pain may be due to

- Stretching of nerve endings in the periosteum covering osteophytes.
- May arise from micro fractures in subchondral bone.
- Stretching of joint capsule due to joint instability.
- Stretching of ligaments.
- Synovitis.
- Capsular fibrosis.

Stiffness:

May be present in morning after a period of inactivity but usually lasts for few minutes. But later it becomes constant and progressive.

Tenderness and Swelling:

- Tenderness usually in the tibial medial condylar area.
- Swelling may be intermittent (in effusion) continuous (in capsule thickening or large osteophytes)
- Restriction of motion
- **Bony crepitus** is characteristic with motion
- Affected joint may be warmth and red
- **Periarticular muscle atrophy** may be due to disease or reflex inhibition of muscle contraction.
- Osteophytes can cause palpable and often visible bony prominences.
- **In advanced stages** gross deformity.

  Bow leg deformity – varus - in medial compartment of knee. Knock knee deformity - valgus - in lateral compartment of knee.
- **In later stage**, joint instability may be due to any of the 3 reasons.
  - Loss of cartilage and bone
  - asymmetrical capsular contracture
  - muscle weakness
- If underlying bone is weak, osteonecrosis

Examination of knee joint

Examination	Normal person	Knee Osteoarthritis patient
Inspection **l.Skin** a. Colour	Normal skin colour Absent	Redness due to inflammation (if acute) Present due to swelling
2. Swelling	Absent	Present around the joint line.
3.Deformity	Absent	Typical varus deformity may be present due to marked tibio-femoral osteoarthritis.
4. Gait	Normal Gait	A jerky, asymmetric 'antalgic' gait due to pain.
5. Muscle wasting	Absent	Quadriceps muscle wasting may be present.

CAUSES OF JOINT PAIN IN PATIENTS WITH OSTEOARTHRITIS.

Source	**Mechanism**
Synovium	Inflammation
Subchondral bone	Medullary hypertension, micro fractures
Osteophyte	Stretching of periosteal nerve endings
Ligaments	Stretch
Capsule	Inflammation, distention
Muscle	Spasm

Examination	Normal person	Knee Osteoarthritis patient
Palpation 1. Warmth	Absent	Present due to inflammation
2.Effusion a.Patellar tap test b.Massage test	Absent Negative Negative	Present Positive in moderate size effusion Positive in small effusion
3. Tenderness	Absent	Joint line or periarticular tenderness present (Tibio femoral joint line, patello femoral joint)
4. Crepitation	Absent	Present due to rough articular surface.
5.Bony enlargement	Absent	Osteophytes may be palpable.
6. Nodules	Absent	Loose bodies may be felt.
7. Measurements of muscle girth (10cm above the patella)	Normal	Quadriceps muscle wasting may be present.
8. Movements a. Extension b. Extensor lag c. Flexion (Measure the range of active flexion using a goniometer)	 Normal Absent Normal range of movement (0-140 degrees)	 May be restricted Present due to quadriceps muscle weakness Flexion deformity present (15-110 degrees)

Laboratory and Radiographic findings:

The diagnosis of osteoarthritis is usually based on clinical and radiographic features. In the early stages, the radiograph may be normal, but joint space narrowing becomes evident, as articular cartilage is lost. A plain radiograph is the only useful investigation. This may show one or more of the typical features of osteoarthritis. They are

1. Focal narrowing of joint space
2. Marginal osteophvte
3. Subchondral sclerosis
4. Osteochondral ("loose") bodies
5. Deformity
6. Chondrocalcinosis

Radiographic features:

Characteristic findings:

- Asymmetric loss of cartilage (narrowing of joint space)
- Sclerosis of subchondral bone under the area of cartilage loss.
- Cyst close to articular surface
- Osteophytes at the margin of the joints
- Osteochondral loose bodies as intra articular bony fragments

Others:

- Old fracture which leads to osteoarthritis
- Evidence of congenital defects
- Chondrocalcinosis
- Later stage displacement of joints
- If severe, bone destruction,total knee replacement is the line of treatment.
- **Radio isotope bone scans** shows discrete increased uptake in osteoarthritic joints due to bone remodeling.

CT and MRI:

May be helpful in evaluating the early stages of degenerative joint disease. But, they are rarely necessary for establishing the diagnosis.

Laboratory Findings:

- Regarding osteoarthritis laboratory findings are not diagnostic. But, specific laboratory test helps to know the underlying causes of secondary osteoarthritis.

- Analysis of synovial fluid reveals mild leucocytosis (<2000 WBC/ microlitre) with a predominance of mononuclear cells .Analysis of synovial fluid is helpful in diagnosing various types of arthritis by changes in its viscosity, cell content and biochemical features.

Prior to the appearance of radiographic changes, the ability to diagnose osteoarthritis clinically without an invasive procedure (eg. Arhtroscopy) is limited. Approaches such as magnetic resonance imaging (MRI) and ultrasonography have not been sufficiently validated to justify their routine clinical use for diagnosis of osteoarthritis or monitoring of disease progression.

Differntial diagnosis:

A number of conditions may mimic Osteoarthritis in knee. They are

1. Rheumatoid arthritis:

It is an autoimmune disorder. It commonly affects women between 24-40 years. Characteristically it is bilaterally symmetrical. Initially it affects the small joints of hand or foot and may spread to large joints. Painful swelling of the joints with stiffness and deformity may occur. Muscle spasm and muscle wasting may be present. Restriction of movement is common. X-ray shows decalcification and diminished joint space may be seen. RA factor is mostly positive.

Gonococcal arthritis:

It occurs 3 weeks after the primary infection. Onset is sudden, with fever. It mostly affects knee or elbow. Pain, redness, heat and edema occur in the affected joint. Gonococci may be demonstrated in urethral discharge.

Tuberculous arthritis:

Tuberculosis of the knee is more common in children than adults; Pain, swelling and wasting of thigh muscles are commonly seen. The knee feels warm. Movements are restricted and often painful. The Mantoux test is positive and the erythrocyte sedimentation rate may be increased. X-ray shows generalised decalcification, obliteration of joint space with erosion of the articular ends.

Septic arthritis:

Acute pyogenic infection of the knee is common. The organism is usually Staphylococcus aureus. The joint is swollen, painful and inflamed. The white

blood cell count and erythrocyte sedimentation rate are elevated. Aspiration reveals pus in the joint.

Syphilitic arthritis:

It is also common in knee. The joint is swollen, painless and shows hypermobility.

Avascular necrosis:

Idiopathic necrosis causes joint pain and local effusion. Early on the diagnosis is made by MRI. Once bone destruction occurs the X-ray changes can be mistaken for those of Osteoarthritis; The cardinal distinguishing features is that in osteonecrosis the 'joint space' (articular cartilage) is preserved in phase of progressive bone collapse and deformity.

Inflammatory Arthropathy

RA, Ankylosing spondylitis and Reiter's disease may start in one or two large joints. X-rays show a predominantly atrophic or erosive arthritis.

DISH : Diffuse Idiopathic Skeletal Hyperostosis

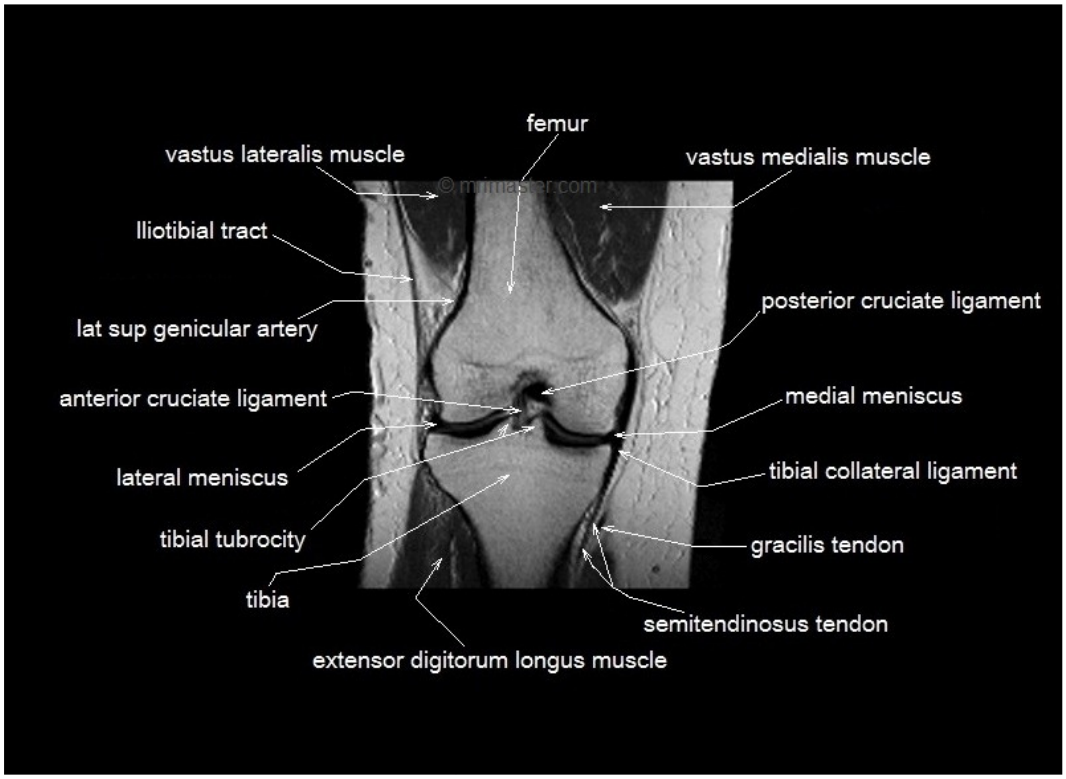
This is a fairly common disorder of middle aged people, characterised by bone proliferation at the ligament and tendon insertions around peripheral joints and the intervertebral discs. On X-ray examination the large bony spurs are easily mistaken for osteophytes.

Complications:

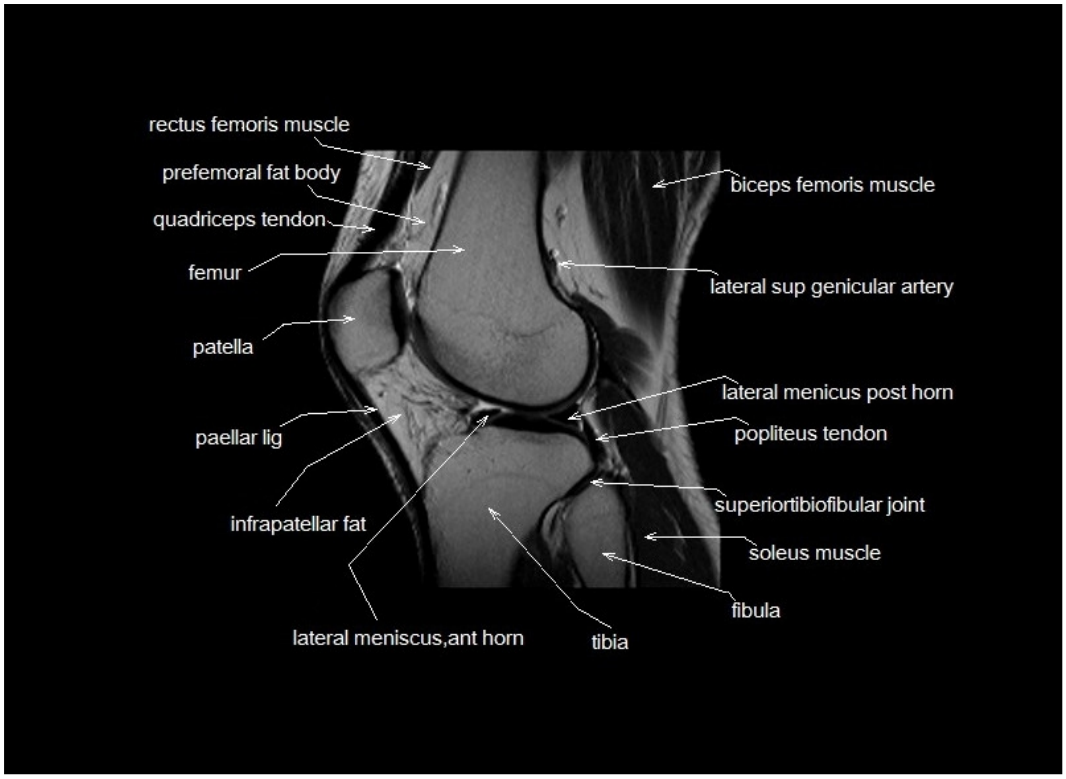
- Bone Death (Osteonecrosis)
- Stress fractures
- Capsular herniation
- Loose bodies
- Rotator cuff dysfunction
- Spinal canal stenosis
- Spondylolisthesis

KNEE JOINT MRI

Sagittal Section

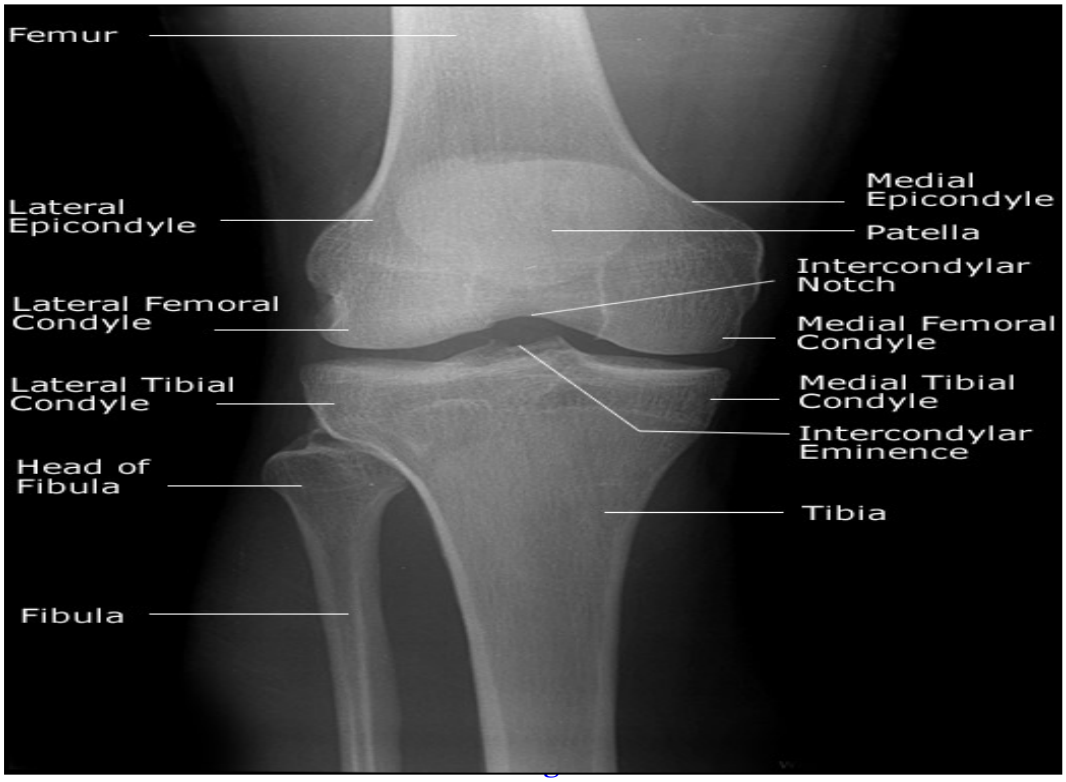


Sagittal Section

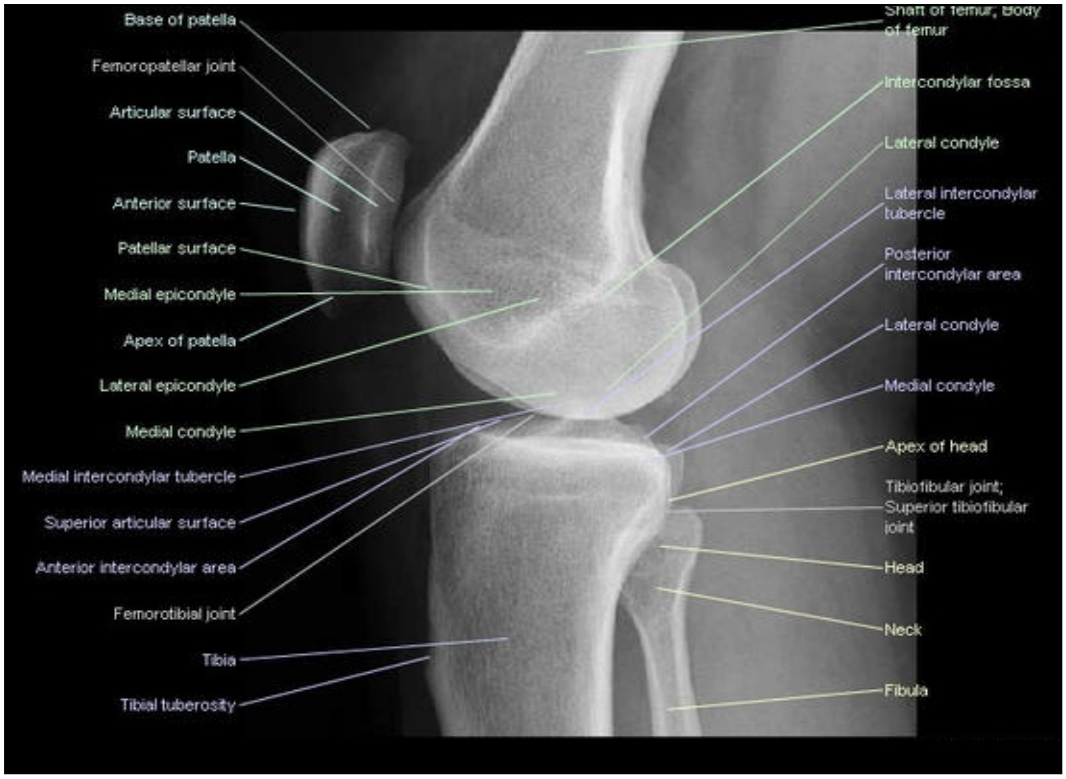


KNEE - Right

Radiograph – AP View



Radiograph – Lateral View



Diagnosis:

Presence of asymmetric knee joint involvement, with pain, tibio femoral, patello femoral tenderness, with or without deformity (varus more than valgus) and the presence of patello femoral crepitus clinically confirms the diagnosis. Confirmatory diagnosis is made by radiological findings.

MATERIALS AND METHODS

The study of **Azhal Keel Vayu** was carried out at Post Graduate Department of **Pothu Maruthuvam**, **Government Siddha Medical College and Hospital, Palayamkottai** under the observation and guidance of the author's Head of the Department. Out of the 40 cases treated, 20 cases were selected in the In Patient and rest 20 in the Out Patient ward. The cases were selected from the Post-graduate Out Patient Department (Pothu-Maruthuvam) according to the signs and symptoms mentioned in the Siddha Maruthuvam text book.

Selection of the cases:

For this clinical study 20 patients of both sexes and varying age groups suffering from **Azhal Keel Vayu** were selected and admitted in the In Patient ward of the **Government Siddha Medical College and Hospital, Palayamkottai** .

Aetiological Factors:

The seasonal variations and precipitating factors like emotional stress, trauma, occupation and climatic change were enquired. The socio economic status, family history and other significant diseases already treated, were thoroughly studied.

Selection Criteria:

- Affection of any one of the major joints with or without swelling.
- Tenderness
- Restricted movements.
- Crepitation present in the affected joints.

In Siddha system the following aspects were taken into consideration.

- Poriyal Arithal
- Pulanal Arithal
- Vinathal
- Examination of Uyir Thathukal
- Ennvagai thervugal
- Udal Kattugalin Nilaigal
- Neerkuri, Neikuri

The following investigations were done in Modern medicine aspect.

HAEMATOLOGICAL INVESTIGATIONS:

a) Total WBC count.
b) Differential WBC Count.
c) Erythrocyte Sedimentation Rate.
d) Haemoglobin percentage.
e) Blood Sugar
f) Blood Urea
g) Serum chlolesterol.

URINE ANALYSIS:

a) Albumin.
b) Sugar.
c) Deposits.

STOOL EXAMINATIONS:

a) Ova.
b) Cyst

SPECIAL INVESTIGATIONS:

X-ray of the affected joint, AP Lateral view

Selection of the drugs:

Selection of the drug was made after a profound study of various Siddha literatures.

The test drug:

Poduthalai Chooranam as Internal Medicine

PODUTHALAI CHOORANAM:

2gm twice a day, morning and night, with hotwater after food

The drug selected for the study was subjected to Pharmacological and Bio-chemical analysis.

Pharmacological analysis was done at the Department of Pharmacology, and Bio-chemical analysis was done at the Department of Biochemistry, Govt. Siddha Medical College, Palayamkottai.

All the patients admitted for the study were given uniformly regular hospital diet.

During discharge all the patients were advised to attend the Out Patient ward at Post Graduate Department of **Pothu Maruthuvam , Government Siddha Medical College & Hospital, Palayamkottai** for further follow-up.

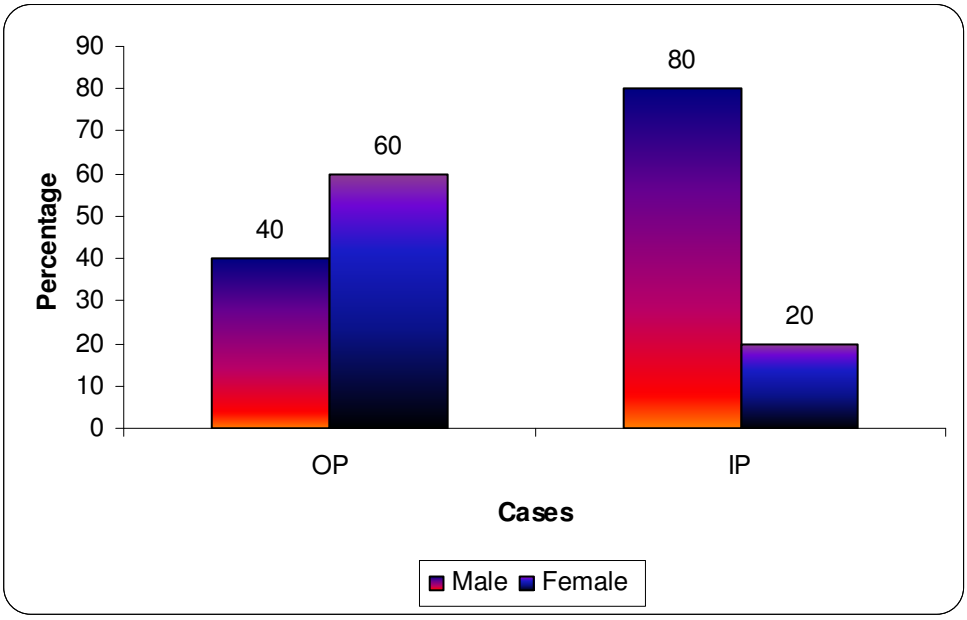
OBSERVATION AND RESULTS

The results were observed regarding the following criteria by clinical study on 40 patients of 20 Inpatients and 20 Out patients.

1. Sex Distribution
2. Age Distribution
3. Kaalam
4. Constitution of body
5. Gunam
6. Religion
7. Paruvakaalam
8. Thinai
9. Socio-economic status
10. Precipitating factors
11. Food habits
12. Occupational status
13. Clinical Manifestations
14. Duration of Disease
15. Kanmenthiriyam
16. Mukkutram
    a. Derangement of vatham
    b. Derangement of Pitham
    c. Derangement of Kabam
17. Ezhu udal kattugal
18. Envagai Thervugal
19. Neer Kuri
20. Nei Kuri
21. Gradation of Results.

1. SEX DISTRIBUTION

Sl. No	Sex	No. of Cases		Percentage %	
		OP	IP	OP	IP
1.	Male	8	16	40	80
2.	Female	12	4	60	20

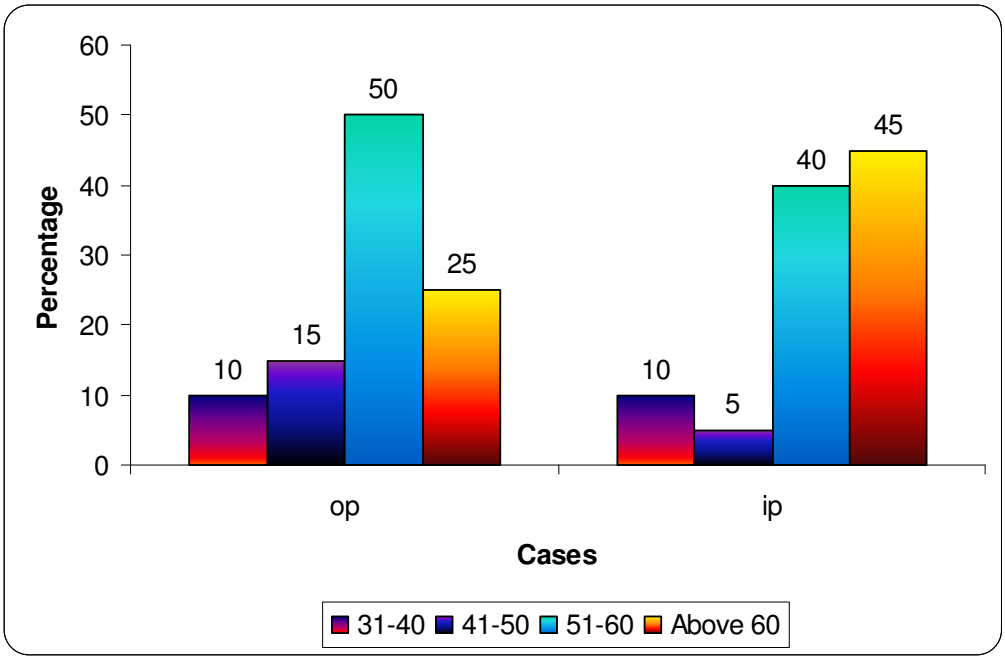


Inference :

Among 40 cases, the prevalence of the disease was found to be higher in Male (80%).

2. AGE DISTRIBUTION

Age groups in years	No. of .cases		Percentage %	
	OP	IP	OP	IP
31-40	2	2	10	10
41-50	3	1	15	5
51-60	10	8	50	40
Above 60	5	9	25	45
Total	20	20	100	100

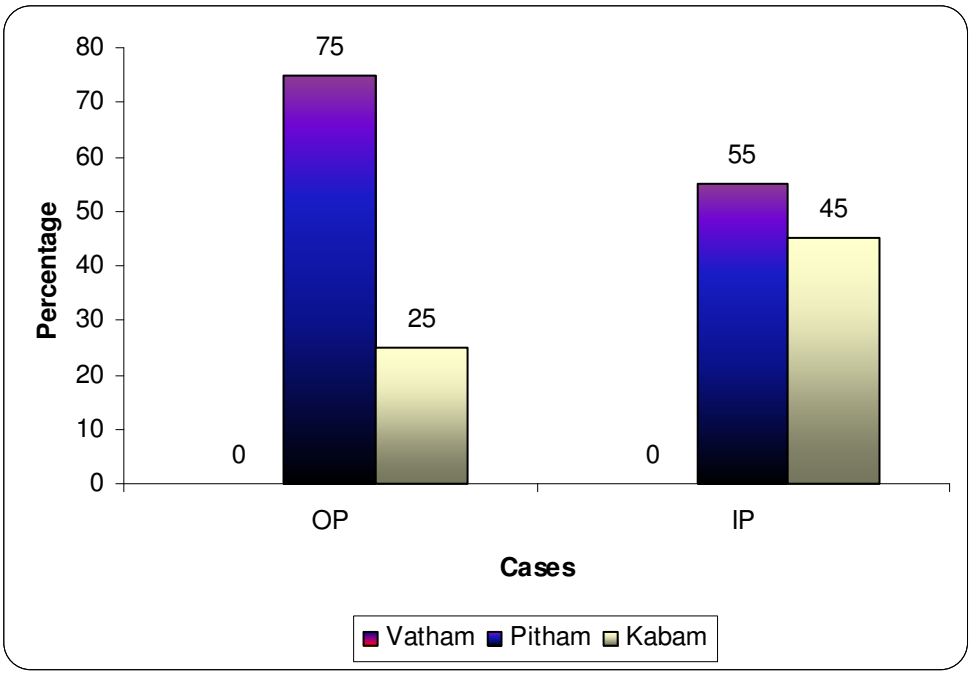


Inference :

The incidence of osteo arthritis was found to be higher among the age group above 51 - 60 .

3. MUKKUTRA KAALAM

Sl. No	Kaalam	No. of cases		Percentage %	
		OP	IP	OP	IP
1.	Vatham	0	0	0	0
2.	Pitham	6	13	30	65
3.	Kabam	14	7	70	35

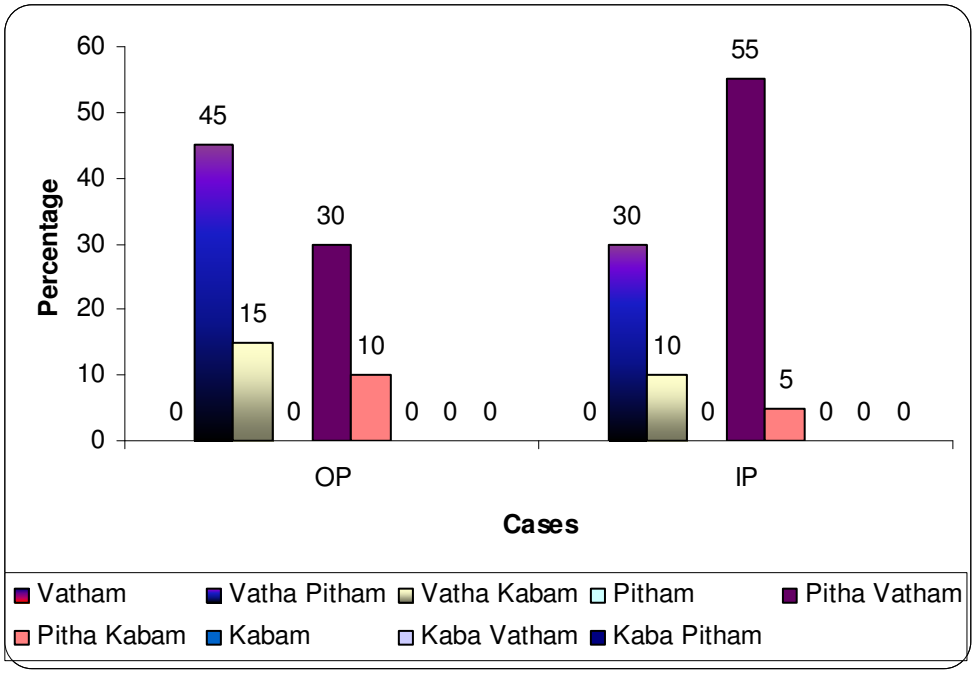


Inference :

Incidence of Osteo arthritis was reported to be higher in Kaba Kaalam.

4. CONSTITUTION OF BODY

Sl. No	Constitution of body	No. of cases		Percentage%	
		OP	IP	OP	IP
1.	Vatham	0	0	0	0
2	Vatha Pitham	9	6	45	30
3.	Vatha Kabam	3	2	15	10
4.	Pitham	0	0	0	0
5.	Pitha Vatham	6	11	30	55
6.	Pitha Kabam	2	1	10	5
7.	Kabam	0	0	0	0
8.	Kaba Vatham	0	0	0	0
9.	Kaba Pitham	0	0	0	0

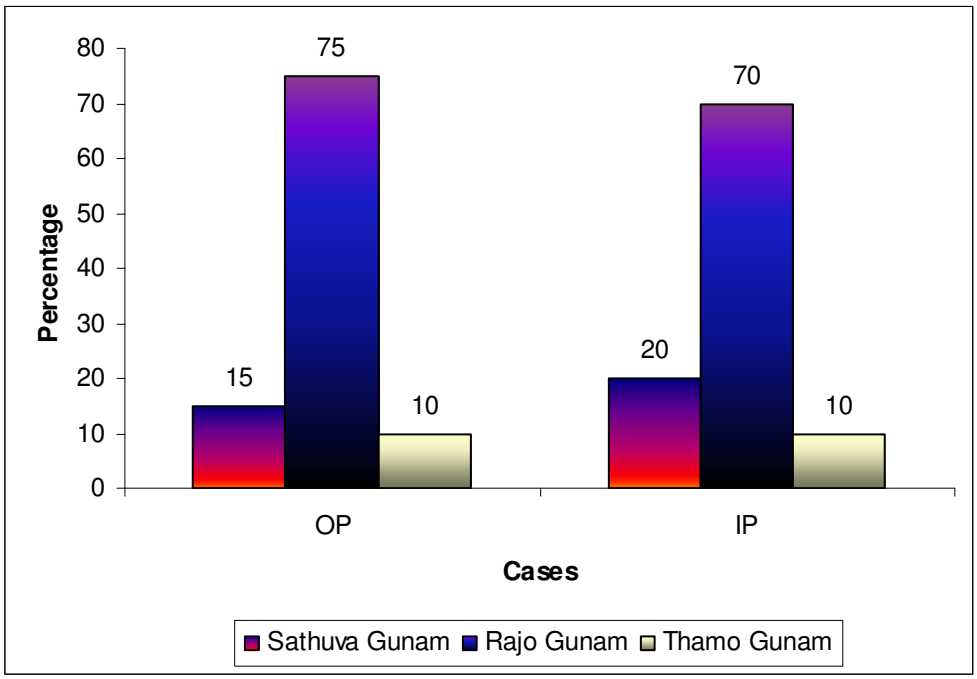


Inference :

Pitha vatha thegis registered high incidence of Osteo arthritis.

5. GUNAM

Sl. No.	Gunam	No. of cases		Percentage %	
		OP	IP	OP	IP
1.	Sathuva Gunam	3	4	15	20
2.	Rajo Gunam	15	14	75	70
3.	Thamo Gunam	2	2	10	10

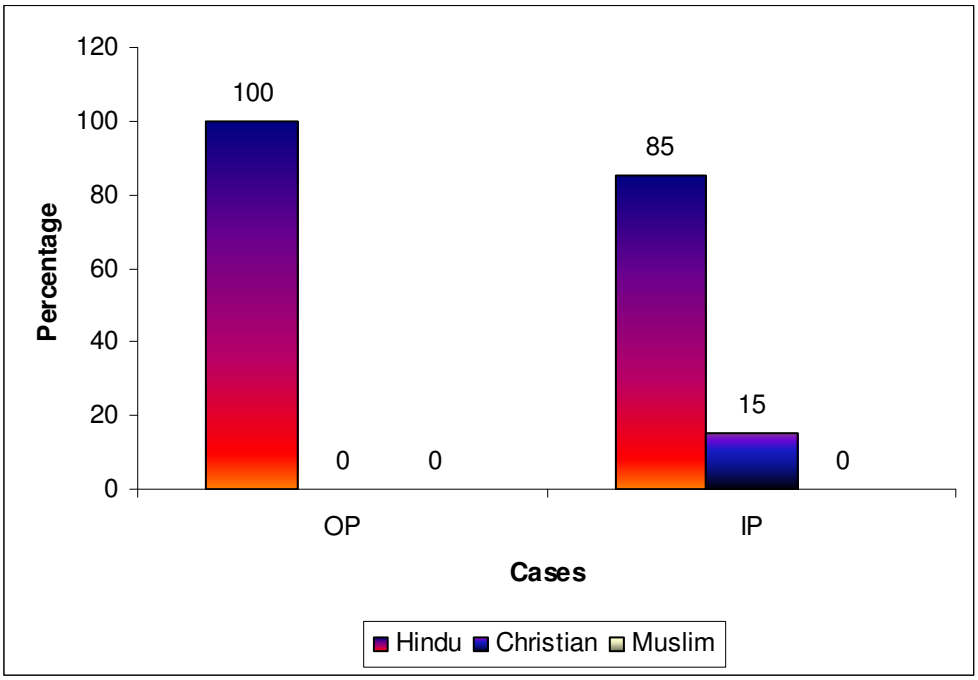


Inference :

In both op and Ip study, cent percentage belongs to Rajo Gunam.

6. RELIGION

Sl. No.	Religion	No. of. Cases		Percentage%	
		OP	IP	OP	IP
1.	Hindu	20	17	100	85
2.	Christian	0	3	0	15
3.	Muslim	0	0	0	0

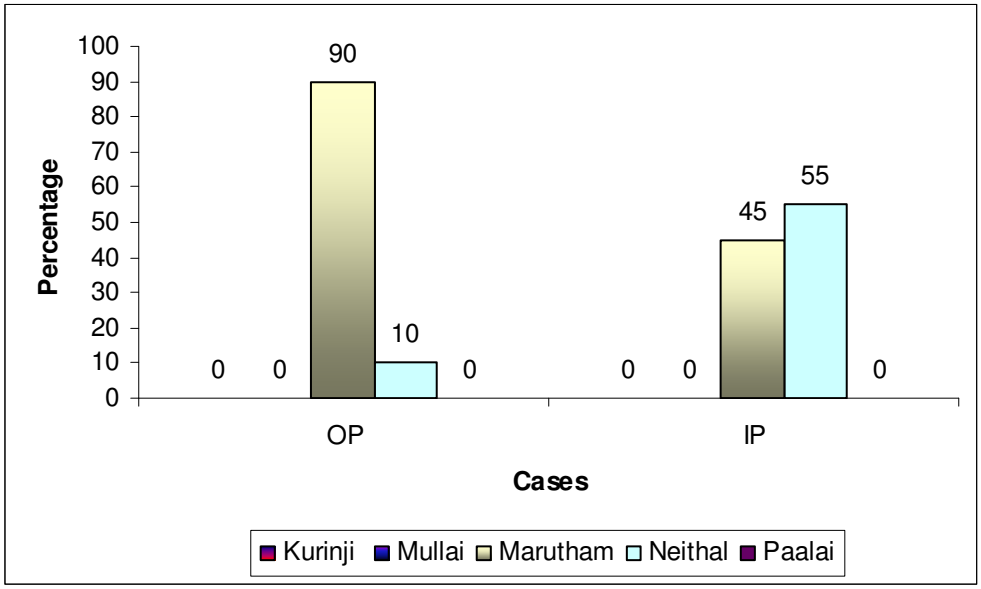


Inference :

Osteo arthritis occured more among the Hindus.

7. THINAI.

Sl. No.	Thinai	No. of Cases		Percentage%	
		OP	IP	OP	IP
1.	Kurinji	0	0	0	0
2.	Mullai	0	0	0	0
3.	Marutham	18	9	90	45
4.	Neithal	2	11	10	55
5.	Paalai	0	0	0	0

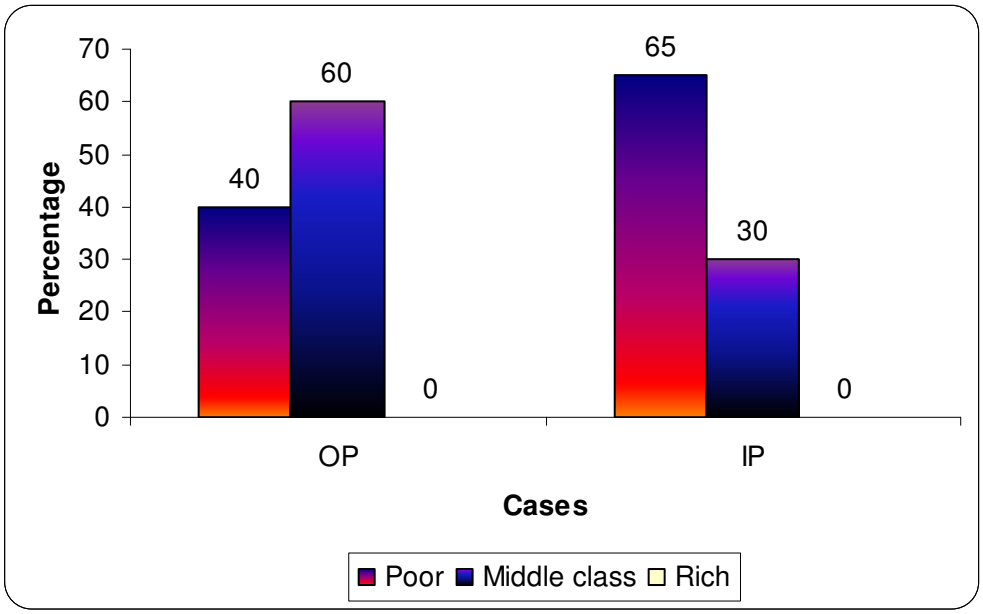


Inference :

Marutham was the place of highest incidence of the disease.

8. SOCIO-ECONOMIC STATUS.

Sl. No.	Socio- Economic Status	No. of cases		Percentage%	
		OP	IP	OP	IP
1.	Poor	8	13	40	65
2.	Middle class	12	7	60	30
3.	Rich	0	0	0	0

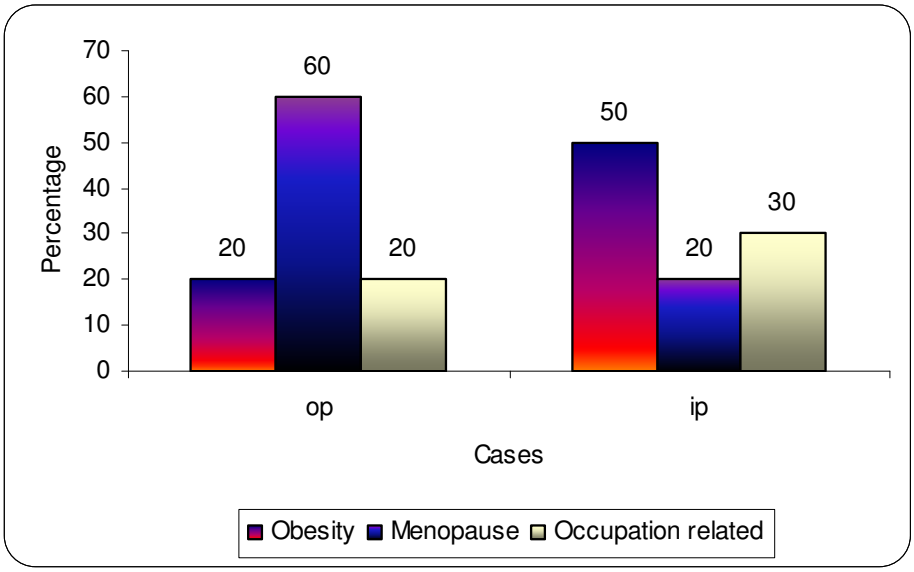


Inference :

Prevalence of the disease was found to be higher in lower economic groups

9. PRECIPITATING FACTORS

Precipitating factors.	No. of cases		Percentage%	
	OP	IP	OP	IP
Obesity	4	10	20	50
Menopause	12	4	60	20
Occupation related	4	6	20	30
Total	20	20	100	100

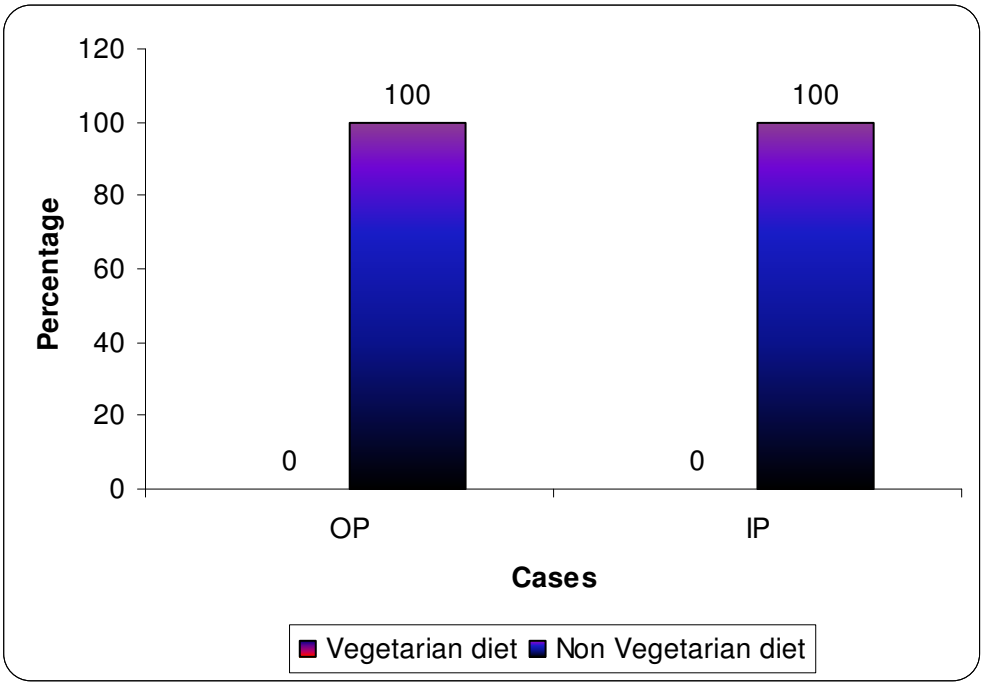


Inference :

Among 40 patients, majority were post menopausal women, 50% were obese and 30%were affected by occupation.

10. FOOD HABITS

Food habits	No. of cases		Percentage%	
	OP	IP	OP	IP
Vegetarian diet	0	0	0	0
Non Vegetarian diet	20	20	100	100
Total	20	20	100	100

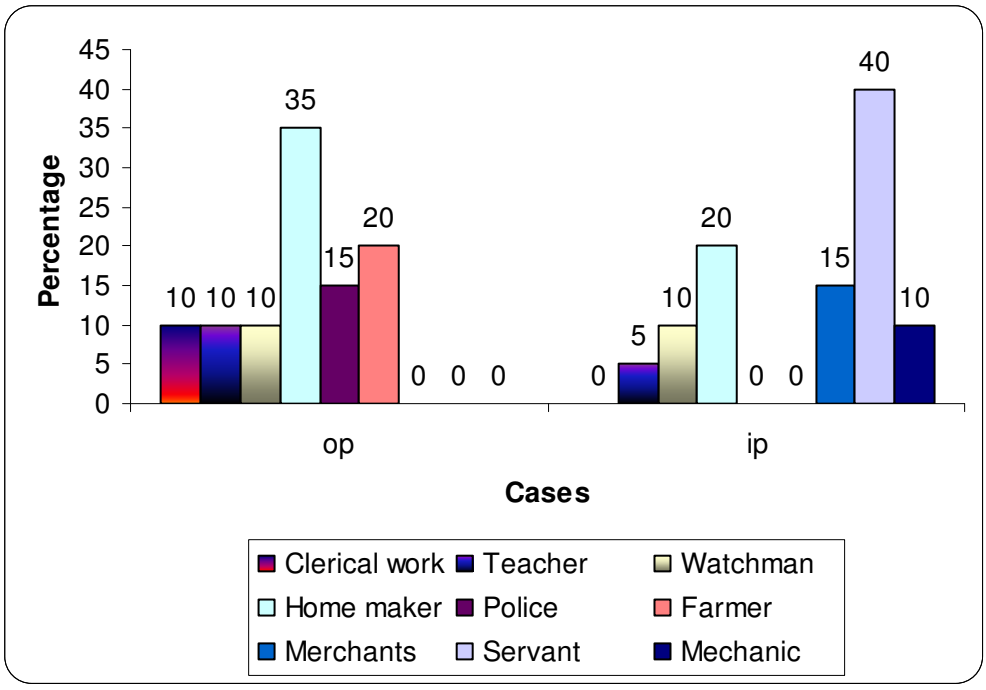


Inference :

Out of 40 cases, 100% of cases were non vegetarians.

11. OCCUPATIONAL STATUS

Occupational Status	No. of cases		Percentage%	
	OP	IP	OP	IP
Clerical work	2	0	10	0
Teacher	2	1	10	5
Watchman	2	2	10	10
Home maker	7	4	35	20
Police	3	0	15	0
Farmer	4	0	20	0
Merchants	0	3	0	15
Servant	0	8	0	40
Mechanic	0	2	0	10
Total	20	20	100	100

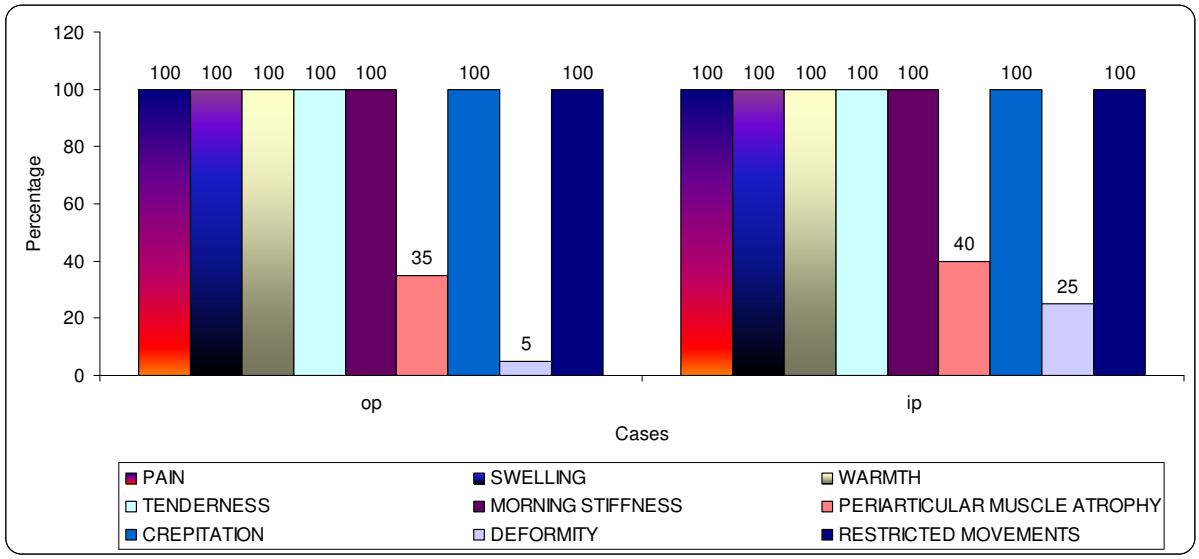


Inference :

Prevalence of the disease was found to be higher among the home makers and farmers.

12. CLINICAL MANIFESTATION:

S. No.	Symptoms	No. of cases		Percentage%	
		OP	IP	OP	IP
1.	PAIN	20	20	100	100
2.	SWELLING	20	20	100	100
3.	WARMTH	20	20	100	100
4.	TENDERNESS	20	20	100	100
5.	MORNING STIFFNESS	20	20	100	100
6.	PERIARTICULAR MUSCLE ATROPHY	7	8	35	40
7.	CREPITATION	20	20	100	100
8.	DEFORMITY	1	5	5	25
9.	RESTRICTED MOVEMENTS	20	20	100	100

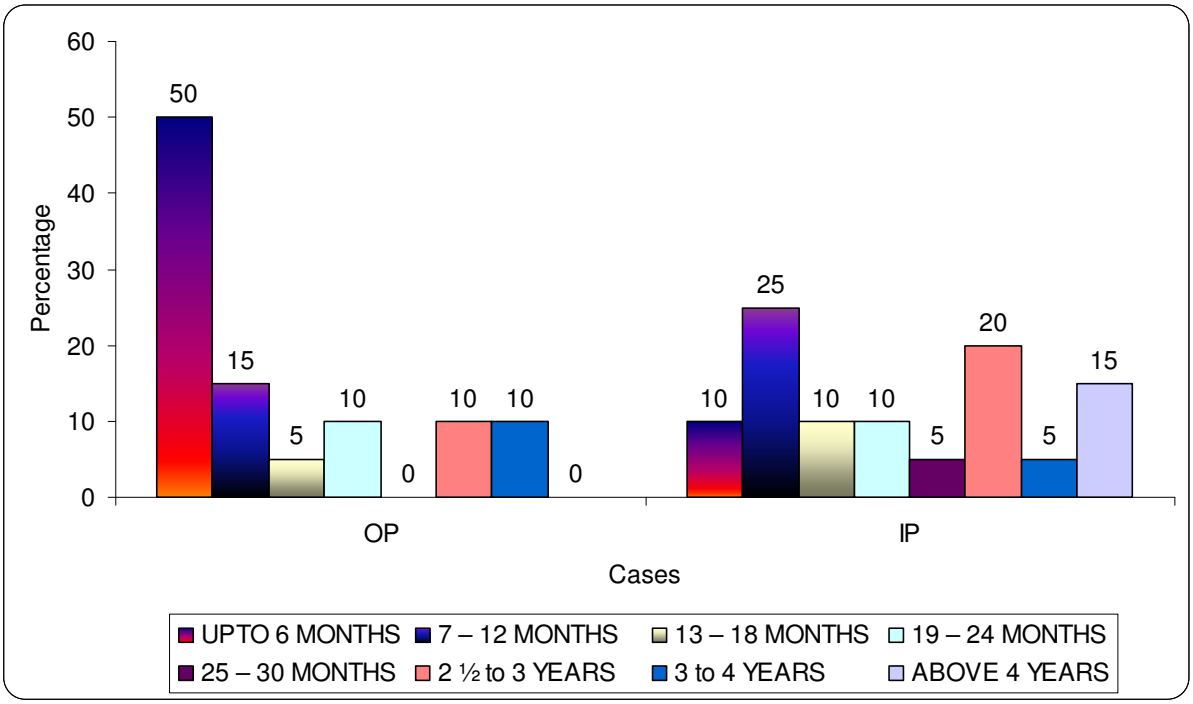


Inference :

Most of the patients had pain, tenderness, crepitation, warmth, swelling and morning stiffness.

13. DURATION OF ILLNESS

Sl. No.	Duration of illness	No. of cases		Percentage%	
		OP	IP	OP	IP
1.	UPTO 6 MONTHS	10	2	50	10
2.	7 – 12 MONTHS	3	5	15	25
3.	13 – 18 MONTHS	1	2	5	10
4.	19 – 24 MONTHS	2	2	10	10
5.	25 – 30 MONTHS	0	1	0	5
6.	2 ½ to 3 YEARS	2	4	10	20
7.	3 to 4 YEARS	2	1	10	5
8.	ABOVE 4 YEARS	0	3	0	15
	TOTAL	20	20	100	100

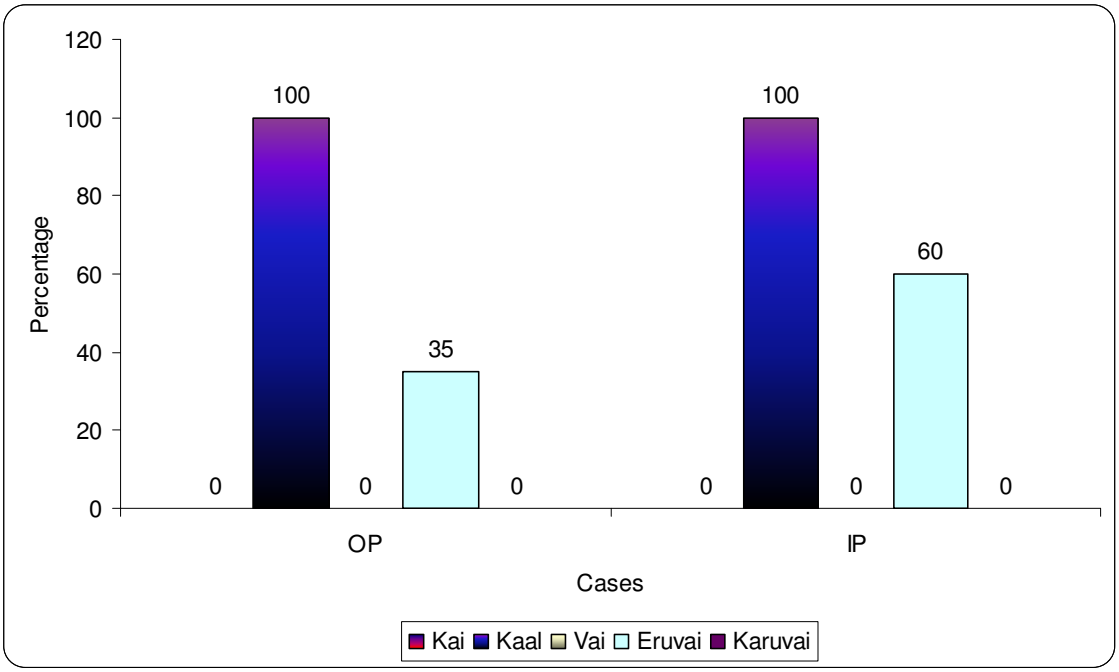


Inference :

Among 40 cases,most of them had the duration of illness more than 6 months.

14. KANMENTHIRIYAM

Sl. No.	Kanmenthiriyam	No. of cases		Percentage%	
		OP	IP	OP	IP
1.	Kai	0	0	0	0
2.	Kaal	20	20	100	100
3.	Vai	0	0	0	0
4.	Eruvai	7	12	35	60
5.	Karuvai	0	0	0	0



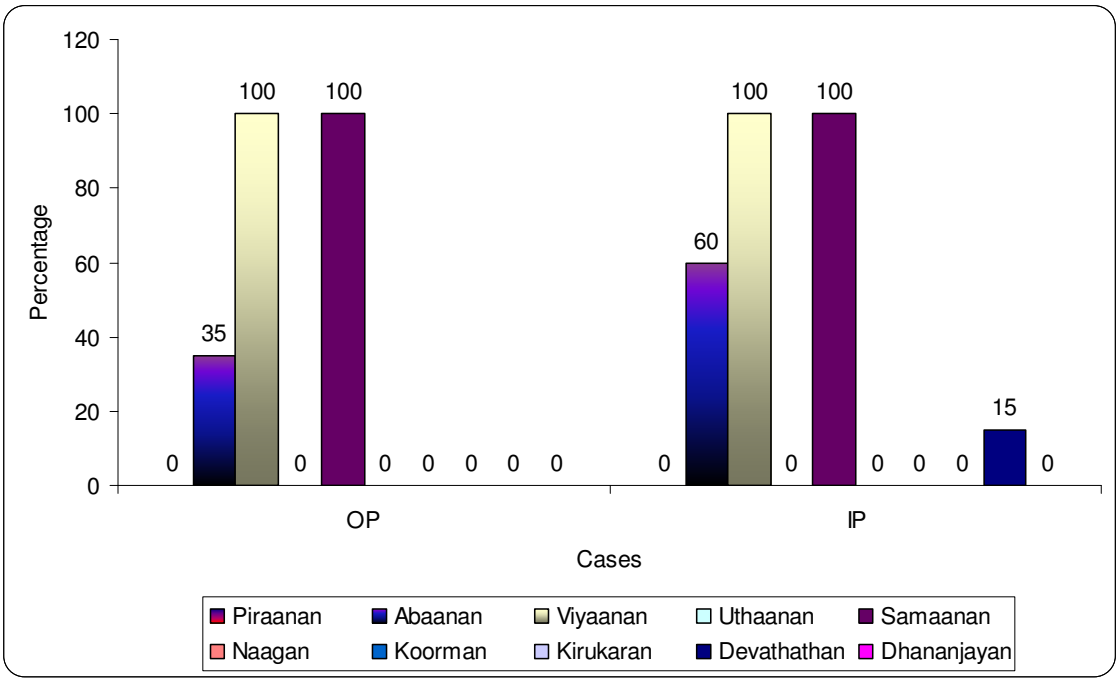
Inference :

Kaal was affected in all cases and Eruvai was affected in majority of the patients (60%).

15. MUKKUTRAM

16.a Derangement of vatham.

Sl. No.	Vatham	No. of cases		Percentage%	
		OP	IP	OP	IP
1.	Piraanan	0	0	0	0
2.	Abaanan	7	12	35	60
3.	Viyaanan	20	20	100	100
4.	Uthaanan	0	0	0	0
5.	Samaanan	20	20	100	100
6.	Naagan	0	0	0	0
7.	Koorman	0	0	0	0
8.	Kirukaran	0	0	0	0
9.	Devathathan	0	3	0	15
10.	Dhananjayan	0	0	0	0

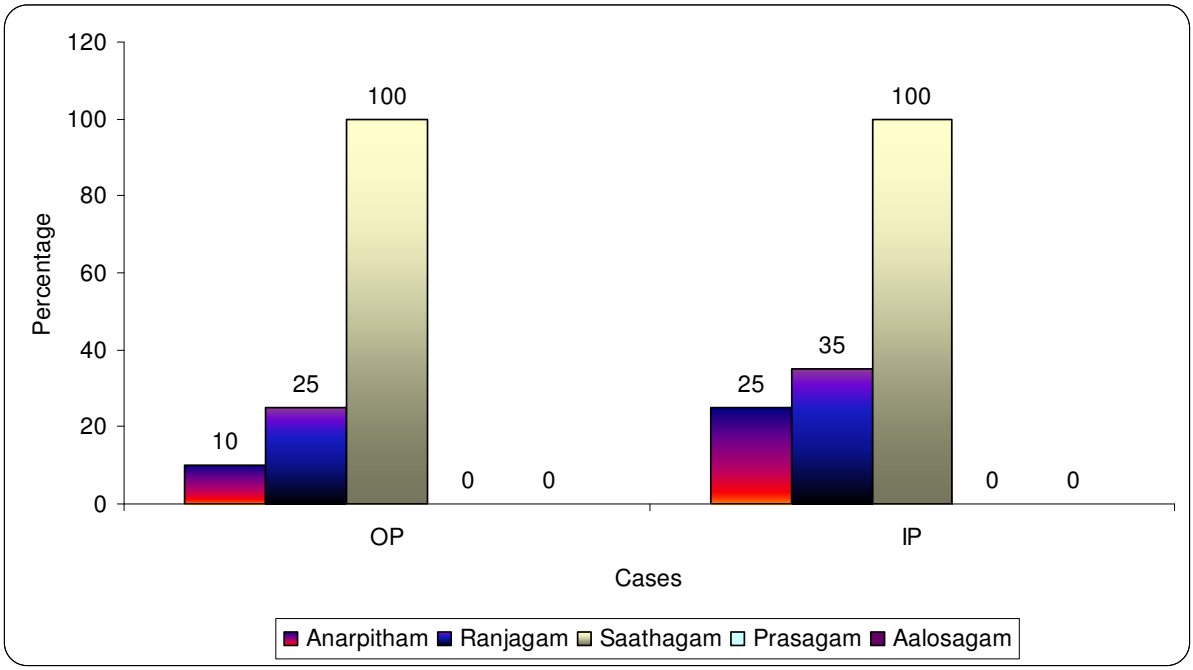


Inference :

Viyaanan,Abaanan & Samaanan were affected in all patients.

16. b. Derangement of pitham.

Sl. No.	Pitham	No. of cases		Percentage%	
		OP	IP	OP	IP
1.	Anarpitham	2	5	10	25
2.	Ranjagam	5	7	25	35
3.	Saathagam	20	20	100	100
4.	Prasagam	0	0	0	0
5.	Aalosagam	0	0	0	0

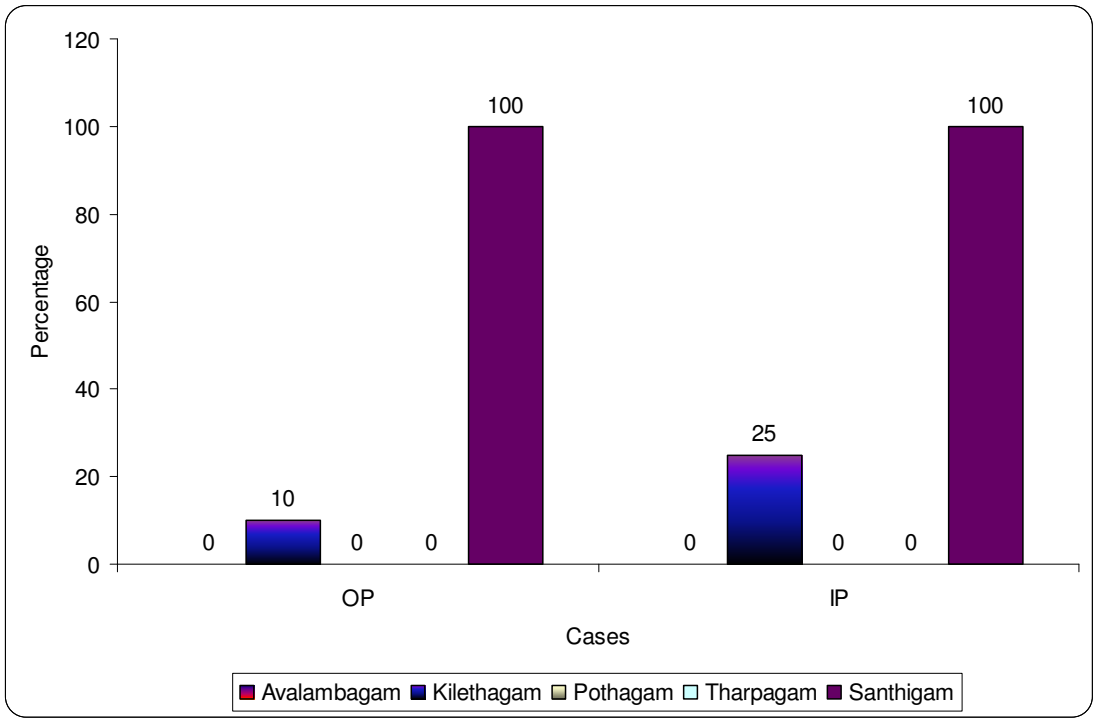


Inference :

Saathagam was affected in all patients with derangement of Anarpitham and Ranjagam in some patients.

16.c. Derangement of Kabam.

Sl. No.	Kabam	No. of cases		Percentage%	
		OP	IP	OP	IP
1.	Avalambagam	0	0	0	0
2.	Kilethagam	2	5	10	25
3.	Pothagam	0	0	0	0
4.	Tharpagam	0	0	0	0
5.	Santhigam	20	20	100	100



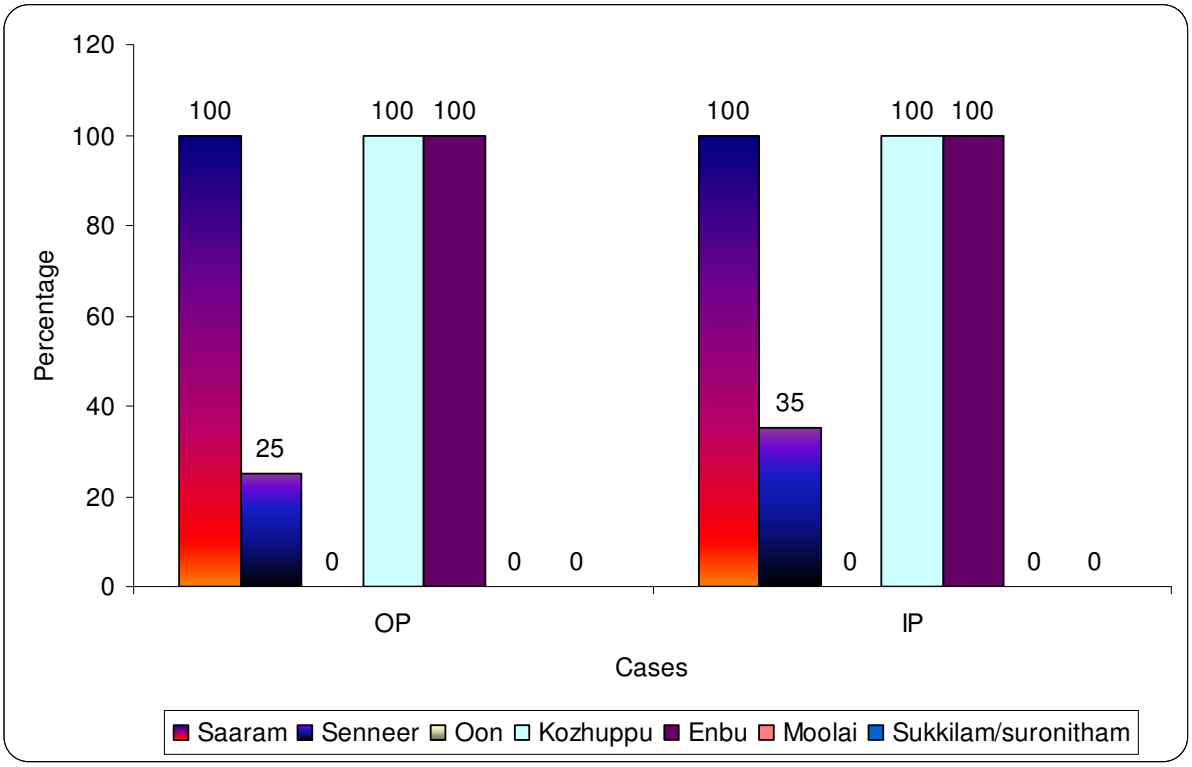
Inference :

Santhigam & Kilethagam were affected in all the patients in this disease.

1

7. Ezhu udal kattugal

Sl. No.	Udal kattugal	No. of cases		Percentage%	
		OP	IP	OP	IP
1.	Saaram	20	20	100	100
2.	Senneer	5	7	25	35
3.	Oon	0	0	0	0
4.	Kozhuppu	20	20	100	100
5.	Enbu	20	20	100	100
6.	Moolai	0	0	0	0
7.	Sukkilam/suronitham	0	0	0	0

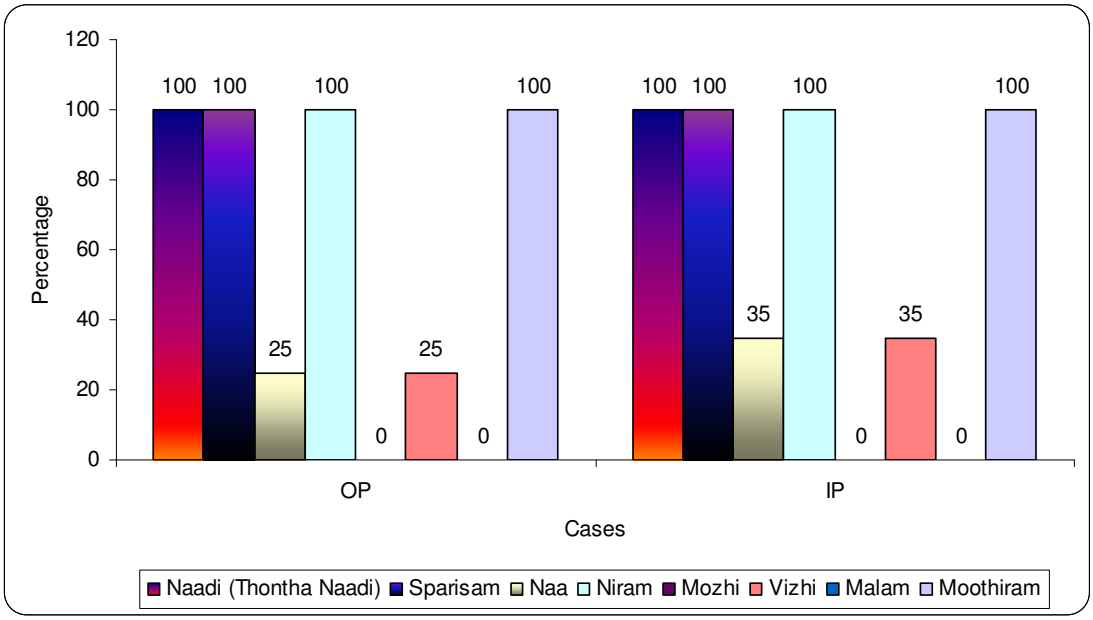


Inference :

Saaram , Kozhuppu and Enbu were affected in all the patients in this disease.

18. **ENVAGAI THERVUAL**

Sl. No.	Envagai thervugal	No. of cases		Percentage%	
		OP	IP	OP	IP
1.	Naadi (Thontha Naadi)	20	20	100	100
2.	Sparisam	20	20	100	100
3.	Naa	5	7	25	35
4.	Niram	20	20	100	100
5.	Mozhi	0	0	0	0
6.	Vizhi	5	7	25	35
7.	Malam	0	0	0	0
8.	Moothiram	20	20	100	100

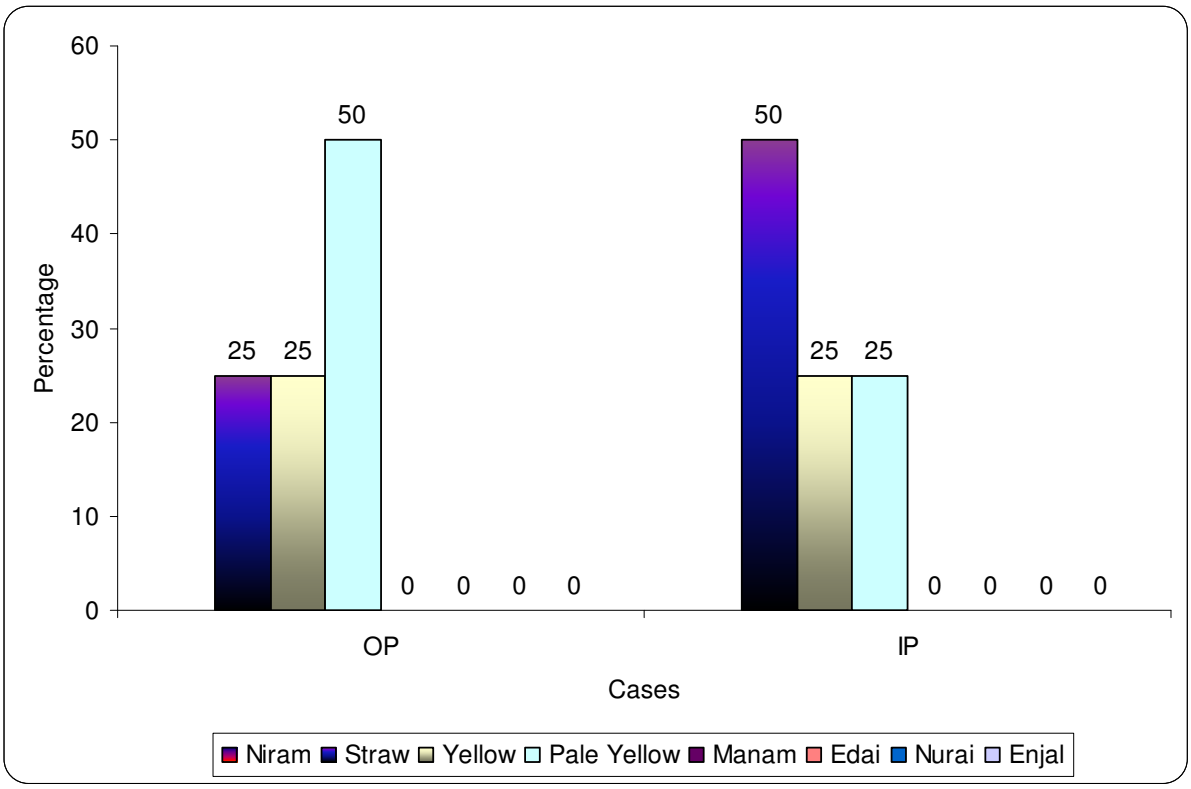


Inference :

Naadi observed in majority of the patients is indicative of azhal keel vayu; Sparisam and Niram were affected in all cases; Vizhi and Naa were affected in 35% of cases.

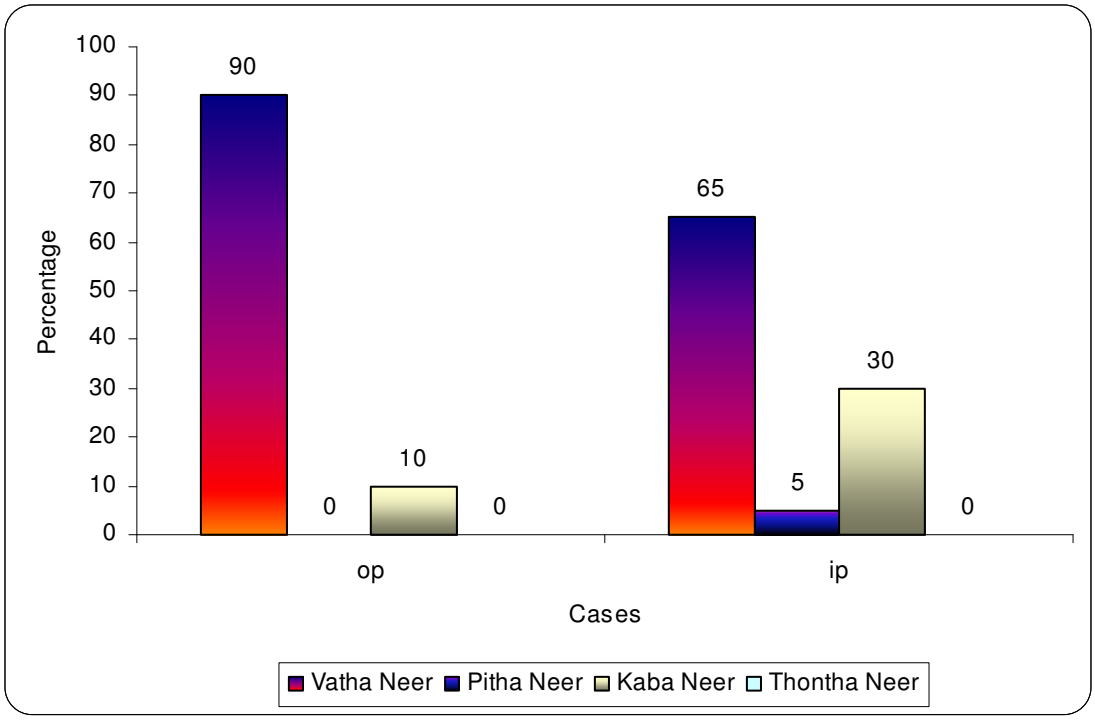
19. **NEER KURI**

Sl. No.	Neer kuri	No. of cases		Percentage%	
		OP	IP	OP	IP
1.	Niram				
	Straw	5	10	25	50
	Yellow	5	5	25	25
	Pale Yellow	10	5	50	25
2.	Manam	0	0	0	0
3.	Edai	0	0	0	0
4.	Nurai	0	0	0	0
5.	Enjal	0	0	0	0



20. NEI KURI

Nei Kuri	No. of cases		Percentage%	
	OP	IP	OP	IP
Vatha Neer	18	13	90	65
Pitha Neer	0	1	0	5
Kaba Neer	2	6	10	30
Thontha Neer	0	0	0	0
Total	20	20	100	100

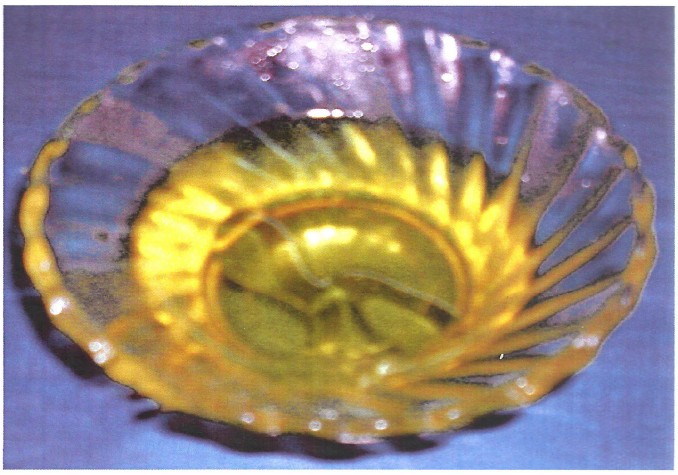


Inference :

Vatha Neer was found in most of the cases.

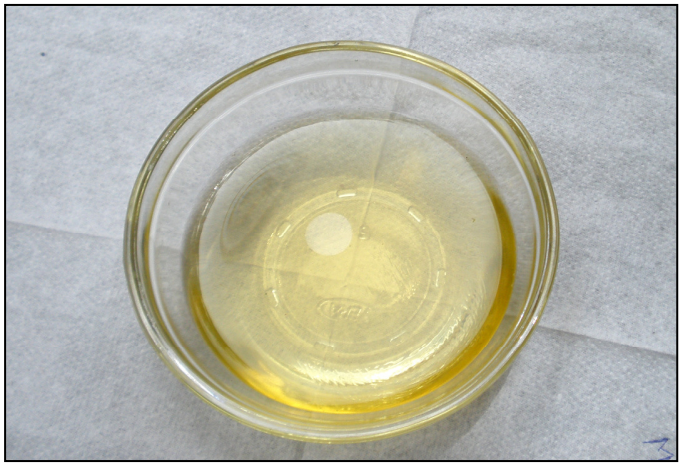
நெய்க்குறி

வாதநீர்



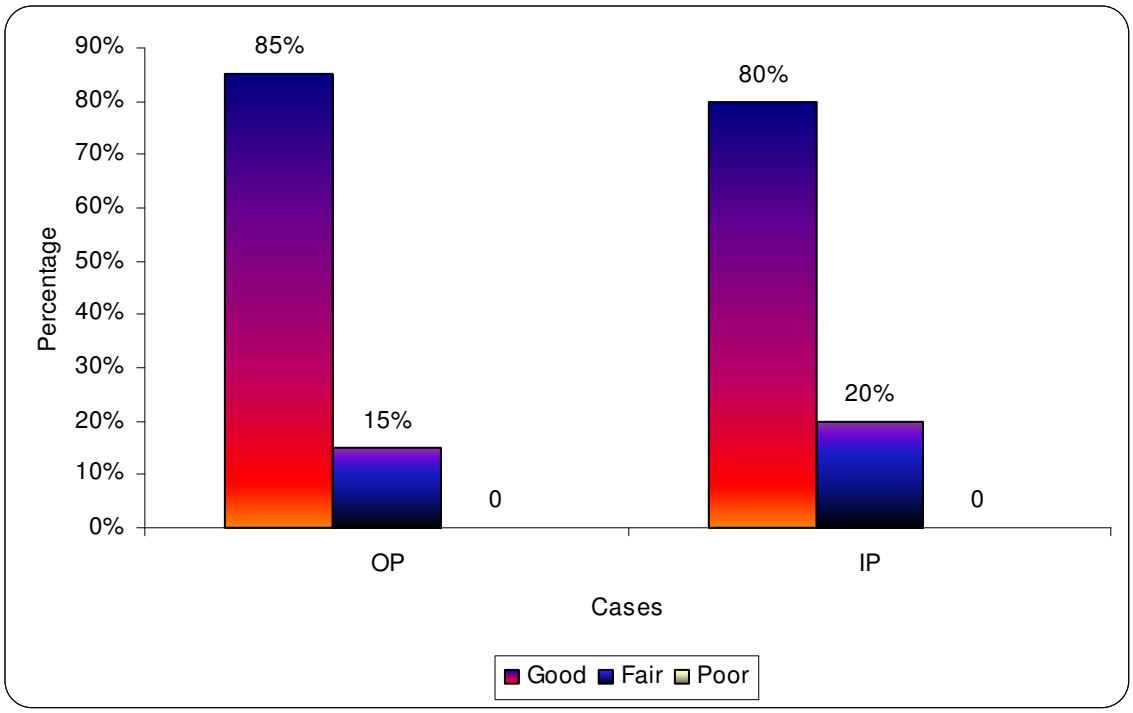
பித்த நீர்

கப நீர்



21. GRADATION OF RESULTS.

Sl. No.	Gradation of Results	No. of cases		Percentage%	
		OP	IP	OP	IP
1.	Good	17	16	85%	80%
2.	Fair	3	4	15%	20%
3.	Poor	-	-	-	-



In op study

85% of cases showed Good response, 15% of cases showed Fair response.

In Ip study

80% of cases showed Good response, 20% of cases showed Fair response.

DISCUSSION

This retrospective review of the disease **Azhal Keel Vayu** begins from the correlation of Azhal Keel Vayu with signs and symptoms of the disease **Osteoarthritis** and then initial sorting the information among the data extracted for the analysis include age, sex, duration of presenting symptoms, predisposing or precipitating factors that provoked attack and their radiological findings. It is clinically identical with Osteo arthritis.

In this study, 20 patients of different age groups and both the sexes were observed as out patient and 20 patients were admitted as inpatient in the Post Graduate Pothumaruthuvam department of Government Siddha Medical College, Palayamkottai, after careful analysis of their clinical symptoms under the supervision of Professor, and Asst.lecturers of Pothu Maruthuvam department.

All the patients were examined on the basis of both siddha as well as modern aspects and all the necessary investigations were made.

All the patients were administered with the trial medicines regularly. The observations were recorded and discussed under the following parameters.

Sex Variation

Among the 20 cases studied as out patients 8 cases were male and 12 cases were female.

Among the 20 cases of In patients 16 cases were male and 4 cases were female.

Majority affected sex is female. The common cause for this may be depletion of calcium from their body, Multiple pregnancies and increased house hold works. From history taking these were concluded as the reasons for female predominance.

Age Variation

Among the 20 cases of out patients, 10% of cases were between 31 – 40 years, 15% of cases were between 41 – 50 years, 50% of cases were between 51 – 60 years and 25% cases were above 60 years.

Among the 20 cases Inpatients, 5% of cases were between 41 – 50 years, 40% of cases were between 51 – 60 years, 45% of cases were between above 60 years.

According to this study, most of the patients were above the age of 60 which was already explained by modern science that degeneration due to ageing is an important cause of OA.

Mukkutra kaalam

Out of 20 cases of out patients 0% of cases were in Vatha kaalam, 75% of cases were in pitha kaalam and 25% of cases were in kaba kaalam.

Out of 20 cases of In patients 55% of cases were in pitha kaalam and 45% of cases were in kaba kaalam.

According to the above observation most of the cases were in Pitha kaalam.

Constitution of the body

In outpatient study 45% of cases belongs to vatha pitha, 15% belongs to vatha kaba thegi, 30% of cases belongs to pitha vatha thegi, 10% of cases belongs to pitha kaba thegi.

In Inpatient study 30% of cases belongs to vatha pitha, 10% belongs to vatha kaba thegi, 55% of cases belongs to pitha vatha thegi, 5% of cases belongs to pitha kaba thegi.

From this observation pitha vatha thegis are registered high incidence of Azhal Keel Vayu .

Gunam

In out patients study 15% of cases belongs to Sathuva Gunam, 75% of patients belongs to Rajo Gunam. 10% of cases belongs to Thamo Gunam.

In In Patients study 20% of cases belongs to Sathuva Gunam, 70% of patients belongs to Rajo Gunam. 10% of cases belongs to Thamo Gunam.

Religion

In out patients study 100% of cases belongs to Hindu religion, 0% of cases belongs to Christian religion and 0% of cases belongs to Muslim religion.

In Inpatients study 85% of cases belong to Hindu religion and 15% of cases belongs to Christian religion and 0% of case belongs to Musilm religion.

Thinai

In Outpatient study 90% were from Marutham and 10% were from Neithal.

In Inpatient study 45% were from Marutham and 55% were from Neithal.

Marutha Nilam is the area where the severity of disease is less but this incidence may be due to alteration in food habits and their activities.

Socio –Economic status.

In outpatient study 40% belongs to poor, 60% belongs to middle class and 0 % belongs to rich.

In Inpatient study 65% belongs to poor and 30% belongs to middle class.

In this study most of the cases belongs to middle class and poor socio economic status. It may due to strain and improper diet habits.

Precipitating factors.

In out patients study 30% of cases were Obese, 35% of cases have attained Menopause, 35% of cases were affected by Occupation.

In Inpatients study 15 % of cases were obese, 80 % of cases have attained Menopause, 5% of cases were affected by Occupation.

Ageing is the most powerful risk factor for Azhal Keel Vayu. Apart from that exposure of joints to repititive occupational use, hormonal inbalance due to menopause and Obesity also plays a significant role in the development of Osteo arthritis.

Food habits.

In outpatients study 100% of patients were non vegetarians.

In Inpatients study 100% of patients were non vegetarians

Diet plays a major role in maintaining ideal body mass index. Majority of the people (90%) were non – vegetarians. Almost all the patients were fond of eating high calorific food like snacks, deep fried items. The calorics consumed exceeds the calories burnt which results in obesity. Out of 20 patients, 8 were overweight .

This was aptly explained by Yugi.

“..... கிழங்கு தன்னை
மிக வருத்தி தயிர்த்தான் கொண்டால்
முகரவே முதுகொலும் மீறியேப்பு முறுக்கி நொந்து
முழங்காலும் கணுக்காலும் கடுப்புண்டாகும்”

- *யூகி சிந்தாமணி*

So Azhal keel vayu may affect both categories of people but predominant among the non vegetarians.

Family history.

In both OP and IP studies cent percentage have no family history.

Occupational status

In out patients study 10% of cases were Clerical workers, 10% of cases were Teachers, 10% of cases were watchmen, 35% of cases were home makers, 15% of cases were police and 20% of cases were farmers.

In Inpatients study 5% of cases were Teachers, 10% of cases were watchmen, 60% of cases were home makers, 15% of cases were merchants, 5% of cases were Servants and 5% of cases were Mechanic.

Occupation plays a important role in the aetiology of Azhal Keel Vayu. House wives and farmers accounts for majority of the cases. The main cause for this is excessive repititive joint loading.

Clinical Manifestation.

In out patients study 100% patients had the complaints of pain, swelling, Warmth, Tenderness, Morning stiffness, Crepitation and Restricted movements.35% of cases had Periarticular Muscle Atrophy, 5% of cases had deformities.

In Inpatients study 100% patients had the complaints of pain, swelling, Warmth, Tenderness, Morning stiffness ,Crepitation and Restricted movements. 40% of cases had Periarticular Muscle Atrophy, 25% of cases had deformities.

Mode of onset

In OP study 100% patients had gradual onset.

In IP study 100% had gradual onset of disease.

Duration of illness.

In OP Study 50% were affected with the duration of Upto 6months, 15% had 7-12 months duration, 5% were 13-18 months duration, 10% were 19 months– 24 months duration, 10% were 2 ½ – 3 years duration and 10% were 3-4 years duration.

In IP Study 10% were affected with the duration of Upto 6months, 25% were 7-12 months duration, 10% were 13-18 months duration, 10% were 19 months– 24 months duration, 5% were 25-30 months duration, 20 % were 2 ½ – 3 years duration and 5% were 3-4 years duration and 15% were above 4 years duration.

According to this study, DOI varies from 6 months -4 years.

Kanmenthiriyam

In OP study 100% had affected kaal and 35% had affected Eruvai.

In IP study 100% had affected kaal and 60% had affected Eruvai.

Uyir thathukkal.

Uyir Thathukkal constitute three vital humours mentioned in siddha system namely Vatham, Pitham and Kabam. Disturbances in Uyir Thathukkal leading to disease entities are discussed here.

i. Disturbances of Vatham.

In OP study 35% of cases had affected abaanan, 100% had affected viyaanan and samaanan.

In IP study 60% of cases had affected abaanan, 100% had affected viyaanan and samaanan and 15% of cases had affected Devathathan.

Most of the patients of Azhal keel Vayu had Constipation because of the defect in Abana vayu. Affected viyaanan produced pain in both knee joints. Affected samaanan produced indigestion. Affected devathathan produced insomnia.

ii. Disturbances of Pitham.

In OP study 10% had affected Anarpitham, 25% had affected Ranjaga pitham and 100% of cases had affected Saathagam.

In IP study 25% had affected Anarpitham, 35% had affected Ranjaga pitham and 100% of cases had affected Saathagam.

Affected Anarpitham produced indigestion. Affected Ranjagam produced Pandu. Affected Saathagam produced difficulty in their normal activities.

iii. Disturbances of kabam

In OP study 10% had affected kilaethagam and 100% of cases had affected Santhigam.

In IP study 25 % had affected kilaethagam and 100% of cases had affected santhigam.

Affected kiletham produced indigestion. Affected santhigam produced joint pain and swelling

Ezhu udal kattugal.

In both OP and IP studies, 100% had affected Saaram, Kozhuppu and Enbu.

In OP study 25% had affected Senneer.

In IP study 35% had affected Senneer.

Affected Saaram produced general debility, affected Senneer produced pandu, affected Oon and Kozhupu produced crepitation and affected Enbu produced joint pain.

Envagai Thervugal

The important Criteria for the diagnosis of the disease is Envagai Thervugal.

All patients had Thontha Naadi and affected sparisam, Niram and Moothiram.

In majority of the cases, the naadi felt was in accordance with the naadi nadai of the disease explained in our siddha literature, sathaga nadi.

Neerkuri

In OP study 25% of cases had straw colour urine, 25% of cases had yellow colour urine and 50 % of cases had Pale yellow colour urine.

In IP study 50% of cases had straw colour urine, 25% of cases had yellow colour urine and 25 % of cases had Pale yellow colour urine.

Neikuri

In OP study 90% had vatha neer and 10% had kaba neer.

In IP study 65% had vatha neer, 5% had pitha neer and 30% had kaba neer

Laboratory Investigations:

There was a remarkable change found in the Laboratory findings. At the same time, total WBC count,Total RBC and Hb were slightly increased after treatment. Blood sugar, Blood Urea and Serum Cholesterol, Creatinine levels showed no significant changes in this study.

The radiographic studies showed narrowed joint space and presence of osteophytes. The trial drug was showed very good improvement and good prognosis of the disease clinically rather than in radiographic changes.

Treatment:

Treatment was aimed at normalising the deranged thodams and providing relief from symptoms. Before treatement, the patients were advised to take vellai Ennai 15 ml with hot water in the early morning for purgation. The patient was adviced to take rest without internal medicine on that day.

Then the Author treated the patient with the trial drug, Poduthalai Chooranam – 2 gm BD with Hot water or Inji Saaru. During treatment, the patients were advised to follow pathiyam (Avoid tamarind, tubers etc.)

Effect of treatment:

- Good improvement was observed in 33 patients
- Moderate improvement was observed in 7 patients
- No toxic and side effects were clinically observed in any case.

Evaluation of Medicine:

Due to the derangement of vatham, constipation was present in many cases at the beginning of the treatment.But during the course of treament the patients showed good relief of their constipation which implies the laxative property of the trial drug.

The Biochemical study of Poduthalai Chooranam reveals the presence of calcium, ferrous iorn, sulphate chloride, phosphate, unsaturated compound and amino acid.

Pharmacological study of the trial drug exhibits significant Anti pyretic activity, Good Analgesic, Good acute and Chronic Anti inflammatory activity.

SUMMARY

1. The clinical study on **Azhal Keel Vayu** with reference to its aetiology, pathogenesis,investigations,clinical features,diagnosis & treatment was conducted at PG Pothu Maruthuvam Department,Government Siddha Medical College, Palayamkottai.
2. Clinical diagnosis of Azhal keel vayu was done on the basis of clinical features described in Sabapathi Manuscript in siddha text book
3. The Aetiology, Pathology, Pathogenesis, Clinical Features, Classification and Prognosis of the disease were collected from a number of literatures both in Siddha system as well as in Modern system of medicine.
4. For this study, 20 patients were diagnosed clinically and admitted in the In patients ward and treated with trial medicines. Another twenty as out patients. Most of the In patients were followed in the out patients department after discharge.
5. The selection of patients and management of patients during admission and after treatment was carried out under the supervision of Professor,Reader and Asst. Lecturer of P.G. Pothu Maruthuvam department.
6. A case sheet proforma was prepared with particular focus on siddha and modern clinical parameters.
7. Separate case sheets were maintained for each patient in In – patient ward and their vital signs & symptoms were monitored and recorded daily . The patients were treated with the trial medicines.
8. Signs and symptoms along with various factors mentioned in the case sheets were elaborately discussed in the previous chapter.
9. Laboratory diagnosis of Azhal Keel Vayu was done by modern scientific methods in the Govt. Siddha Medical College Hospital, Palayamkottai.
10. Routine blood examinations, urine, stools and radiological investigation were also considered for diagnosis and to follow the progress of the patients.
11. Siddha diagnosis was made with help of Ezhu udalkattugal and Envagai Thervugal especially with naadi and neikuri.
12. The trial medicine choosen for internal treatment for the management of Azhal Keelavayu was **Poduthalai Chooranam -2gm BD with hot water after food**

13. Since Azhal Keel Vayu is a chronic disease, it required treatment for minimum twenty days to minimize the severe pain, tenderness and swelling with slight disappearance of the crepitation. And then the patient was advised to continue the treatment as out patient for some period as per their requirements.
14. Though there was appreciable clinical improvement, there was not much remarkable radiographic changes.
15. The patients were observed for a period of 2 months during and after the course of treatment. No signs of complications were reported. Clinically no toxic effects were noticed during the treatment period.
16. The pharmacological evaluation of the Poduthalai Chooranam had showed Good Anti inflammatory, Analgesic and Significant Antipyretic Activity.
17. All the patients were advised to avoid vayu patharthangal which are the precipitatory factors of the disease.
18. Finally the clinical improvement was graded as Relieved and Improved on the basis of symptoms relieved and the results observed during the study.

CONCLUSION

The Most predominant degenerative disorder, Azhal Keel Vayu was studied in all aspects.

All the cases were treated with the trial Medicine, Poduthalai Chooranam which contains many herbal and mineral ingredients.

Treatment was given on the basis of Mukkutra theory.The deranged kutrams were normalised by the trial medicine.

The trial medicine, Poduthalai Chooranam has the taste of kaarpu, Kaippu according to the taste of individual ingredients.

Kaarpu Suvai	-	Has its functions of relieving indigestion, flatulence and constipation.
Kaippu suvai	-	Regulates the vitiation of pitham, lightens the body and maintains good health.
Thuvarpu suvai	-	It cleans the blood and heals the wound

Thus the trial medicine on the basis of its suvai acts as an effective Anti arthritic drug to yield prognosis in Azhal Keel Vayu.

Almost all the cases treated with trial drug have shown remarkable improvements. Further follow up of all the patients showed excellent relief of their symptoms.

Marked clinical improvement	-	33patients
Moderate clinical improvement	-	7 patients

Relief from the symptom was observed within 20 days in moderate cases and within 40 days in long standing cases.

Evaluation of the trial drug was done pharmacologically and exhibited

Significant	-	Antipyretic activity
Good	-	Analgesic activity
Good	-	Acute and chronic anti inflammatory effect

There was no report of Adverse side effects during the entire course of treatment.

Thus the treatment of Azhal Keel Vayu with Poduthalai Chooranam was found clinically very effective and safe.

பொடுதலை

Phyla nodiflora

பொடுதலை சூரணம்

ANNEXURE - I

PREPARATION OF TRIAL DRUG

INTERNAL MEDICINE:

Poduthalai Chooranam (Gunapaadam Mooligai Part 1 Pg. No. 712)

INGREDIENTS:

Poduthalai Samoolam (Phyla Nodiflora)

PURIFICATION OF DRUGS:

PODUTHALAI SAMOOLAM

The whole plant is taken and cleaned completely in water and made it dry.

PREPARATION :

PROCESS:

The raw drug is powdered and kept in an airtight container

DOSAGE : 2gms BD with hot water after food

DURATION : 30 Days

INDICATIONS : Osteo Arthritis

Reference :

"Gunapadam Mooligai – 1st edition Murugesa Mudailiyar, Pg. No: 712

Table

SI. No	Name	Characters	Chemical constituents	Action
1.	**பொடுதலை Phyla Nodiflora Verbenaceae**	சுவை -கைப்பு, துவர்ப்பு தன்மை-வெப்பம் பிரிவு -கார்ப்பு	Essential Volatile oil, Alkaloid Pyrethrin (Pellitorin), Anacyclin, Inulin, Sesamin, Tyramine amides, Hydrocarbolin.	**Anti-inflammatory, Diuretic, Astringent, Expectorant, Tonic**

ANNEXURE II

BIO CHEMICAL ANALYSIS OF PODUTHALAI CHOORANAM

Preparation of the Extract:

5gms of the drug is weighed accurately and placed in a 250ml clean beaker. Then 50ml of distilled water is added to it and dissolved well. Then it is boiled well for about 10 minutes. It is cooled and filtered in a 100ml volumetric flask and then it is made up to 100ml with distilled water. This fluid is taken for analysis.

Qualitative Analysis:

Sl.No.	Experiment	Observation	Inference
1	**TEST FOR CALCIUM:** 2ml of the above prepared extract is taken in a clean test tube. 2ml of 4% Ammonium oxalate solution is added to it.	A white precipitate is formed	**Indicates the presence of Calcium**
2	**TEST FOR SULPHATE:** 2ml of the extract is added to 5% Barium chloride solution.	A white precipitate is formed	**Indicates the presence of Sulphate**
3	**TEST FOR CHLORIDE:** The extract is treated with silver nitrate solution.	No white precipitate is formed	Absence of Chloride
4	**TEST FOR CARBONATE:** The substance is treated with concentrated HCL.	No brisk effervessence is formed	Absence of Carbonate
5	**TEST FOR STARCH:** The extract is added with weak iodine solution.	No blue colour is formed	Absence of Starch
6	**TEST FOR IRON FERRIC:** The extract is treated with glacial acetic acid and potassium ferro cyanide**.**	No blue colour is formed	Absence of Ferric iron.
7	**TEST OF IRON FERROUS:** The extract is treated with concentrated nitric acid and ammonium thio cyanate.	Blood red colour is formed	**Indicates the presence of Ferrous iron**
8	**TEST FOR PHOSPHATE:** The extract is treated with Ammonium Molybdate and concentrated nitric acid.	No yellow precipitate is formed	Absence of Phosphate
9	**TEST FOR ALBUMIN:** The extract is treated with Esbach's reagent.	No yellow precipitate is formed	Absence of Albumin.

10.	**TEST FOR TANNIC ACID:** The extract is treated with ferric chloride.	Blue black precipitate is formed	Indicates the presence of Tannic acid
11	**TEST FOR UNSATURATION:** Potassium permanganate solution is added to the extract**.**	It gets decolourised	**Indicates the presence of Unsaturated compound**
12	**TEST FOR THE REDUCING SUGAR:** 5ml of Benedict's qualitative solution is taken in a test tube and allowed to boil for 2 mts and added 8-10 drops of the extract and again boil it for 2 mts.	No colour change occurs	Absence of Reducing sugar
13	**TEST FOR AMINO ACIDS:** One (or) two drops of the extract is placed on a filter **paper and** dried it well. After drying, 1% Ninhydrin is sprayed over the same and dried well.	violet colour is formed	**Indicates the presence of Amino acid**.
14	**TEST FOR ZINC:** The extract is treated with potassium Ferrocyanide**.**	No white precipitate is formed	Absence of zinc

ANNEXURE III

PHARMACOLOGICAL STUDIES

ANALGESIC ACTIVITY

Analgesic activity of *Poduthalai Chooranam* against acetic acid induced writhing reflux in mice

Analgesic activity of *Poduthalai Chooranam* at a dose of 100 mg/kg and 200 mg/kg was evaluated by acetic acid induced writhing reflux in mice. Painful reactions in animals may be produced by the chemicals such as phenylquionolone, bradykinin etc. like that, acetic acid pain reaction which is characterized as a writhing response. Construction of abdomen, turning of trunk (twist) and extension of hind legs are taken as reaction to chemically induced pain. Analgesics (both narcotic and non-narcotic) inhibit writhing response.

Requirements

Animal	:	Swiss albino mice (20 – 25g) either sex
Drugs and chemicals:		Diclofenac sodium (standard), Acetic acid (1%), *Poduthalai Chooranam*

Method

Treatment protocol

Group- 1	:	Treated as normal control received 10ml/kg of normal saline through orally
Group- 2	:	treated as standard control received 10mg/kg of diclofenac sodium through orally
Group- 3	:	treated as Test-I received 100mg/kg of *Poduthalai Chooranam* with 2 ml of sterile water through orally
Group- 4	:	treated as Test- II received 200mg/kg of *Poduthalai Chooranam* with 2 ml of sterile water through orally

Both dose of *Poduthalai Chooranam* were administered one hour prior to the acetic acid administration. Note the onset on writhing. Record the numbers of

abdominal contractions, trunk twist and extension of hind limbs as well as the number of animals showing such response during a period of 10 minutes were noted.

Statistics

Data are expressed as mean ± SEM; data analyzed by one way ANOVA followed by Dunnet's multiple range tests to determine the significance of the difference between the control group and rats treated with extracts.

* Values were considered significant at $P < 0.01$.

Table - 1

Analgesic activity of *Poduthalai Chooranam* against acetic acid induced writhing reflux in mice

Treatment	Dose (mg/kg)	No. of writhing	% reduction in reaction time
Group I Normal saline	Inject 1%v/v acetic acid 1ml/100g of body weight	35.2±2.6	-
Group II Standard	10mg/kg Diclofenac sodium through orally	7.5±0.7	70.69***
Group III *Poduthalai Chooranam*	100mg/kg administered through orally	12.03 ± 0.91	60.25***
Group IV *Poduthalai Chooranam*	200mg/kg administered through orally	11.93 ± 2.5	69.01***

Values are expressed as mean ± SEM

Values are analysed by one way ANOVA followed by Dunnet's multiple range tests

*** Values were considered significant at $P < 0.001$.

Results

The table values show that analgesic activity of *Poduthalai Chooranam*at a dose of 100mg/kg and 200mg/kg by acetic acid induced writhing reflex. The result reveals that both doses of *Poduthalai Chooranam*possess significant analgesic activity at $P < 0.001$.

ACUTE TOXICITY STUDY

Acute oral toxicity refers to those adverse effects occurring following oral administration of a single dose of a single dose of a substance or multiple doses given within 24 hours. Acute toxic class method (OECD guidelines 423, (2000) was followed to arrive at the maximum safety dose of the drug extracts. Three Wistar strain female albino rats (8-12 weeks old, 180-200g body weight) were used in each group. Single dose (2g/kg) of the *Poduthalai Chooranam* was orally administered to overnight fasted (food but not water withheld) animas while control animals received the vehicle (0.3%w/v CMC). Animals were observed individually after dosing at least once during the first 4 hrs and daily thereafter, for a total of 14days. Body weights of the animals were recorded. The other observations include changes for skin, fur, eyes and mucous membranes, respiratory, circulatory and autonomic and central nervous system and somatomotor activity and behavior pattern. At the end of 14 days, all animals were subjected to gross necropsy.

Statistics

Data are expressed as mean ± SEM; data analysed by one way ANOVA followed by Dunnet's multiple range tests to determine the significance of the difference between the control group and rats treated with test compounds.

* Values were considered significant at $P < 0.5$.

Results

Acute toxicity study

All of the rats fed with the food sample showed normal general behavior, respiratory pattern, cardiovascular signs, motor activities, reflexes and normal change in skin and fur.

Table - 2

Hematological values of *Poduthalai Chooranam*in the acute toxicity study

S. No	Parameter	Control	Sample 2g/kg
1	White blood cells ($x10^3/\mu l$)	9.36±0.54	10.98 ± 0.98
2	Hemoglobin (g/dl)	11.50±0.26	9.98 ± 0.95
3	Mean corpuscular volume	60.45±2.3	58.95 ± 2.27
4	Mean corpuscular hemoglobin conc. (g/dl)	34.56±0.86	28.96 ± 0.49
5	Platelet ($x10^5/\mu l$)	5.60±0.52	4.98 ± 0.91
6	Red blood cell ($x10^6/\mu l$)	3.87±0.24	2.95 ± 0.29

Values are expresses as Mean ± S.E.M.

All groups were treated with oral dose of 2g/kg body weight

No significant different from normal control

Table - 3

Blood chemical values of food sample in the acute toxicity study

S. No	Parameter	Control	Sample 2g/kg
1	Glucose (mg/dl)	148.75±3.96	139.60 ± 2.98
2	BUN(mg/dl)	34.26±1.23	29.59 ± 1.95
3	Creatinine(mg/dl)	0.46±0.06	0.38 ± 0.40
4	Total protein (g/dl)	5.48±0.23	4.98 ± 0.29
5	Albumin (g/dl)	3.49±0.62	4.08 ± 0.15
6	Total bilirubin (mg/dl)	0.26±0.02	0.49 ± 0.01
7	AST (u/l)	141.5±3.76	138.20 ± 1.94
8	ALT (u/l)	86.36±1.75	80.40 ± 0.25
9	ALP (u/l)	75.57±2.16	80.71 ± 0.25

Values are expresses as Mean ± S.E.M.

All groups were treated with oral dose of 2g/kg body weight

No significant different from normal control

Discussion and conclusion

In acute toxicity study for 14 days, at a dose of 2g/kg of *Poduthalai Chooranam*sample were chosen for the experiment. In the aspect of general behaviours, the rats treated with food sample at a single dose had no signs of behavior changes and toxic signs. The treated groups revealed no significant differences in body weight gain. The increase in body weight may have resulted from physiological changes in rats such as metabolism, food and water intake. However, the result from animal health monitoring in the entire period of 14days showed no sign of morbidity and diseases.

The albino Wistar rats were healthy as shown by the normal appearance of general behavior, respiratory pattern, cardiovascular signs, motor activities, reflexes and normal change in skin fur.

With regards to hematological values, most of values in treated groups were normal in comparison with the control group. Significantly, some values were different from those of the control group such as RBC, MCV, MCHC, and platelet. However, such values are within the normal ranges. These variations may have resulted from variation among animal groups (Feldman et al., 2000) (Inala et al., 2002). Therefore, these results suggest that the test drug did not cause hematological or immunological defects in rats.

Furthermore, blood chemical examination was performed in order to evaluate any toxic effects on liver. In this study, the levels of these blood chemical values were minor changes and remained within the normal range (Casley and King, 1980) (Levine, 1995) (Angkhasirisap et al., 2002).

In conclusion, *Poduthalai Chooranam* sample given orally to Wistar rats did not produce toxicities.

ANTI-INFLAMMATORY ACTIVITY

Anti-inflammatory activity of *Poduthalai Chooranam* against Carrageenan induced paw edema in rats

The anti-inflammatory activities of *Poduthalai Chooranam* at a dose of 100mg/kg and 200mg/kg body weight were evaluated using Carrageenan induced paw edema method. The inflammation was readily produced in the form of edema with the help of the irritant such as carrageenan. Carrageenan is a sulphated polysaccharide obtained from sea weed (Rhodophyceae) and when injected cause the release of prostaglandins by the way it produces inflammation and edema.

Requirements

Animal	:	Albino rat (180 – 200g)
Drugs and chemicals:		Diclofenac sodium (standard), carrageenan (1%), *Poduthalai Chooranam* Digital plethysmometer UGO Basile (Italy)

Method

The animals were divided into 4 groups each having six animals

Treatment protocol

Group- 1	:	Treated as normal control received 10ml/kg of normal saline through orally
Group- 2	:	Treated as standard control received 10mg/kg of diclofenac sodium through orally
Group- 3	:	Treated as treatment control received 100mg/kg of *Poduthalai Chooranam* with 2 ml of sterile water through orally
Group- 4	:	Treated as treatment control received 200mg/kg of *Poduthalai Chooranam* with 2 ml of sterile water administered through orally

A freshly prepared suspension of carrageenan (1% w/v, 0.1ml) was injected to the plantar region of left hind paw of each rat. One group was kept as control and the animals of the other groups were pretreated with *Poduthalai Chooranam* given through orally 60 min before the carrageenan treatment. The paw volumes of the test compounds, standard and control groups were measured at 60, 120, 180 minutes of carrageenan treatment with the help of Digital plethysmometer UGO Basile (Italy). Mean increase in paw volume was measured and the percentage of inhibition was calculated.

% anti-inflammatory activity = (Vc – Vt/Vc) x 100

Where Vt is mean increase in paw volume in rats treated with test compounds

Vc is mean increase in paw volume in control group of rats

Statistics

Data are expressed as mean ± SEM; data analysed by one way ANOVA followed by Dunnet's multiple range tests to determine the significance of the difference between the control group and rats treated with test compounds.

* Values were considered significant at $P < 0.01$.

Anti-inflammatory activity of *Poduthalai Chooranam* against Carrageenan induced paw edema in rats

Treatment	Dose (mg/kg)	Paw volume(ml) as measured at 3 hour	Percentage inhibition of paw edema
Group I Normal saline	5ml/kg orally	4.95 ± 0.76	
Group II Standard	10mg/kg Diclofenac sodium through orally	1.48 ± 0.26	59.02**
Group III *Poduthalai Chooranam*	100mg/kg administered through orally	2.51 ± 0.01	38.05**
Group IV *Poduthalai Chooranam*	200mg/kg administered through orally	2.95 ± 0.21	52.10**

Values are expressed as mean ± SEM

Values are analyzed by one way ANOVA followed by Dunnet's multiple range tests, to determine the significance of the difference between the control group and rats treated with the test compounds.

** Values were considered significant at $P < 0.01$.

Results

Poduthalai Chooranam at a dose of 100 and 200mg/kg were tested for their anti-inflammatory activity by using carrageenan induced rat paw edema method and the results are tabulated in table. The results reveals that both doses of ***Poduthalai Chooranam*** 100 and 200mg/kg possess significant anti-inflammatory activity when compared to control group at $p<0.01$.

LABORATORY INVESTIGATION OF IN PATIENTS

S.No	IP. NO	TC		DC								ESR				Bl.Sugar		Bl.Urea	
				N		L		E		M		BT		AT					
		BT	AT	BT	AT	BT	AT	BT	AT	BT	AT	1/2 hr	1 hr	1/2 hr	1 hr	BT	AT	BT	AT
1	1051	8500	8400	64	60	33	37	3	3	-	-	20	42	6	12	92	96	30	28
2	1050	8900	9000	66	60	30	38	4	2	-	-	8	16	4	8	215	180	34	30
3	1256	8500	8400	64	58	34	40	2	2	-	-	25	55	8	10	74	88	22	22
4	1283	8400	8500	72	65	29	33	2	2	-	-	5	10	2	4	89	86	28	28
5	694	8500	8500	60	56	37	42	3	2	-	-	22	50	8	10	388	200	25	22
6	3348	9200	9100	70	62	28	36	2	2	-	-	7	15	4	8	78	84	29	28
7	1435	9600	9500	64	60	29	37	7	3	-	-	7	16	2	4	310	250	16	16
8	1411	7500	7600	73	66	25	22	2	2	-	-	2	5	2	4	129	110	20	24
9	1256	9000	9100	63	58	35	40	2	2	-	-	33	68	8	16	87	84	16	20
10	845	8700	8600	65	60	32	38	3	2	-	-	7	18	2	6	249	200	35	35
11	135	8700	8800	63	58	33	40	4	2	-	-	3	7	2	4	97	92	22	22
12	69	7200	7200	49	58	48	39	3	3	-	-	10	20	5	10	78	84	33	33
13	26	8200	8200	58	62	40	36	2	2	-	-	15	30	2	4	95	95	24	26
14	1138	8700	8700	68	62	28	35	4	3	-	-	20	40	6	12	87	86	18	20
15	500	7900	8000	65	60	32	37	3	3	-	-	3	7	2	4	124	100	12	14
16	3554	7900	8100	58	65	40	33	2	2	-	-	3	6	2	4	107	98	26	24
17	777	7200	7300	68	64	32	32	4	2	-	2	10	20	4	10	92	98	28	25
18	900	10200	10300	54	50	28	40	18	8	-	2	2	5	2	4	98	92	24	22
19	3294	8500	8500	68	60	26	36	6	4	-	-	10	20	6	12	53	80	20	22
20	3348	7900	8000	65	60	32	37	3	3	-	-	3	6	2	4	83	90	22	24

LABORATORY INVESTIGATION OF OUT PATIENTS

S.No	OP. NO	TC		DC								ESR				Bl.Sugar		Bl.Urea	
				N		L		E		M		BT		AT					
		BT	AT	BT	AT	BT	AT	BT	AT	BT	AT	1/2 hr	1 hr	1/2 hr	1 hr	BT	AT	BT	AT
1	14176	7300	7400	69	68	28	30	3	2	-	-	10	22	6	12	84	90	19	19
2	14178	9000	9000	52	54	38	40	10	6	-	-	16	20	9	14	174	150	25	24
3	14179	8900	9100	52	57	38	37	10	6	-	-	10	20	6	12	81	88	48	38
4	15681	8100	8200	56	51	40	47	4	2	-	-	10	22	4	10	130	110	27	25
5	15682	7900	7800	67	63	30	35	3	2	-	-	10	4	6	10	80	80	30	28
6	17161	8200	8400	61	64	35	34	4	2	-	-	2	6	2	4	62	80	22	22
7	17185	7300	7300	68	64	30	34	2	2	-	-	3	3	2	4	133	120	31	29
8	18114	8000	8000	68	64	30	34	2	2	-	-	1	7	1	3	83	98	26	24
9	18116	7800	8000	57	60	38	37	5	3	-	-	3	7	2	4	64	88	31	30
10	18806	7200	7400	57	55	38	43	5	2	-	-	3		2	4	86	86	32	32
11	18919	8700	8800	65	62	32	34	3	4	-	-	3	7	2	4	119	100	24	25
12	19193	8100	8200	68	64	30	34	2	1	-	-	5	12	3	7	135	130	22	22
13	19195	7900	8000	59	65	38	32	3	3	-	-	2	5	2	4	243	220	36	35
14	19536	9000	9000	65	60	30	37	5	3	-	-	4	8	2	4	79	88	29	28
15	19619	9300	9500	49	47	48	52	3	1	-	-	4	20	2	4	138	112	32	30
16	19622	8700	9000	62	58	35	39	3	3	-	-	10	24	4	8	228	180	25	25
17	19623	8200	8400	62	58	36	38	2	4	-	2	12	30	4	8	70	82	31	30
18	19627	8100	8200	65	60	31	36	4	4	-	2	15	20	2	4	185	160	28	26
19	19535	7400	7600	68	64	32	34	2	2	-	-	10	17	4	8	156	140	15	16
20	19625	8300	8400	65	60	32	38	3	2	-	-	7	20	3	7	82	96	22	20

LABORATORY INVESTIGATION OF IN PATIENTS

S.No	IP NO	Before treatment				After treatment				Before treatment			After treatment		
				Deposits				Deposits				Occult			Occult
		Albumin	Sugar	Pus Cells	Epi Cells	Albumin	Sugar	Pus Cells	Epi Cells	Ova	Cyst	Blood	Ova	Cyst	blood
1	1051	Nil	Nil	3-5	Nil	Nil	Nil	1-2	Nil	Nil	Nil	-ve	Nil	Nil	-ve
2	1050	Nil	Nil	2-3	Nil	Nil	Nil	1-2	Nil	Nil	Nil	-ve	Nil	Nil	-ve
3	1256	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	-ve	Nil	Nil	-ve
4	1283	Nil	Nil	Nil	1-3	Nil	Nil	Nil	1-2	Nil	Nil	-ve	Nil	Nil	-ve
5	694	Nil	Nil	2-3	Nil	Nil	Nil	1-2	Nil	Nil	Nil	-ve	Nil	Nil	-ve
6	3348	Trace	Nil	4-5	3-5	Nil	Nil	1-2	1-2	Nil	Nil	-ve	Nil	Nil	-ve
7	1435	Trace	Nil	3-5	Nil	Nil	Nil	1-2	Nil	Nil	Nil	-ve	Nil	Nil	-ve
8	1411	Nil	Nil	Nil	1-2	Nil	Nil	Nil	0-1	Nil	Nil	-ve	Nil	Nil	-ve
9	1256	Trace	Nil	3-5	Nil	Nil	Nil	1-2	Nil	Nil	Nil	-ve	Nil	Nil	-ve
10	845	Nil	Nil	1-2	Nil	Nil	Nil	1-2	Nil	Nil	Nil	-ve	Nil	Nil	-ve
11	135	Trace	Nil	1-2	Nil	Nil	Nil	1-2	Nil	Nil	Nil	-ve	Nil	Nil	-ve
12	69	Nil	++	3-5	2-3	Nil	Nil	Nil	1-2	Nil	Nil	-ve	Nil	Nil	-ve
13	26	Nil	Nil	Nil	Nil	Nil	Nil	1-2	Nil	Nil	Nil	-ve	Nil	Nil	-ve
14	1138	Nil	Nil	Nil	1-2	Nil	Nil	Nil	1-2	Nil	Nil	-ve	Nil	Nil	-ve
15	500	Nil	++++	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	-ve	Nil	Nil	-ve
16	3554	Nil	Nil	Nil	5-7	Nil	Nil	Nil	1-2	Nil	Nil	-ve	Nil	Nil	-ve
17	777	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	-ve	Nil	Nil	-ve
18	900	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	-ve	Nil	Nil	-ve
19	3294	Trace	Nil	1-3	Nil	Nil	Nil	Nil	1-2	Nil	Nil	-ve	Nil	Nil	-ve
20	3348	Trace	+	3-5	2-3	Nil	Nil	1-2	1-2	Nil	Nil	-ve	Nil	Nil	-ve

LABORATORY INVESTIGATION OF OUT PATIENTS

S.No	OP. NO	Before treatment				After treatment				Before treatment			After treatment		
				Deposits				Deposits				Occult			Occult
		Albumin	Sugar	Pus Cells	Epi Cells	Albumin	Sugar	Pus Cells	Epi Cells	Ova	Cyst	Blood	Ova	Cyst	blood
1	14176	Trace	Nil	2-4	Nil	Nil	Nil	1-2	Nil	Nil	Nil	-ve	Nil	Nil	-ve
2	14178	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	-ve	Nil	Nil	-ve
3	14179	Nil	+	Nil	1-2	Nil	Nil	Nil	1-2	Nil	Nil	-ve	Nil	Nil	-ve
4	15681	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	-ve	Nil	Nil	-ve
5	15682	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	-ve	Nil	Nil	-ve
6	17161	Nil	Nil	1-2	Nil	Nil	Nil	1-2	Nil	Nil	Nil	-ve	Nil	Nil	-ve
7	17185	Nil	Nil	2-4	Nil	Nil	Nil	1-2	Nil	Nil	Nil	-ve	Nil	Nil	-ve
8	18114	Nil	Nil	Nil	2-3	Nil	Nil	Nil	0-1	Nil	Nil	-ve	Nil	Nil	-ve
9	18116	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	-ve	Nil	Nil	-ve
10	18806	Nil	Nil	Nil	1-3	Nil	Nil	Nil	0-1	Nil	Nil	-ve	Nil	Nil	-ve
11	18919	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	-ve	Nil	Nil	-ve
12	19193	Nil	Nil	2-5	3-4	Nil	Nil	1-2	1-2	Nil	Nil	-ve	Nil	Nil	-ve
13	19195	Trace	Nil	1-2	Nil	Nil	Nil	1-2	Nil	Nil	Nil	-ve	Nil	Nil	-ve
14	19536	Nil	Nil	Nil	1-2	Nil	Nil	Nil	1-2	Nil	Nil	-ve	Nil	Nil	-ve
15	19619	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	-ve	Nil	Nil	-ve
16	19622	Nil	Nil	Nil	1-2	Nil	Nil	1-2	1-2	Nil	Nil	-ve	Nil	Nil	-ve
17	19623	Nil	Nil	1-2	Nil	Nil	Nil	1-2	Nil	Nil	Nil	-ve	Nil	Nil	-ve
18	19627	Nil	Nil	1-2	Nil	Nil	Nil	1-2	Nil	Nil	Nil	-ve	Nil	Nil	-ve
19	19535	Trace	Nil	2-6	1-4	Nil	Nil	1-2	1-2	Nil	Nil	-ve	Nil	Nil	-ve
20	19625	Nil	Nil	4-5	Nil	Nil	Nil	1-2	Nil	Nil	Nil	-ve	Nil	Nil	-ve

LABORATORY INVESTIGATION OF OUT PATIENTS

S.No	OP.No	Name	Hb gm%		Serum Cholesterol mg%	
			BT	AT	BT	AT
1	14176	Alagammal	11	11	256	220
2	14178	Annammal	10.4	10.4	159	160
3	14179	Anbalagan	10.4	11.2	209	186
4	15681	Annammal	10.5	11	178	170
5	15682	Poongothai	10.4	10.6	120	120
6	17161	Chandrasekar	10.8	11.4	200	192
7	17185	Shanmugam	12	12.2	149	146
8	18114	Boomadevi	8.9	10.6	123	120
9	18116	Ponnammal	10.6	10.6	158	160
10	18806	Isakkiammal	10.2	10.8	140	140
11	18919	Prakasi	8.8	9.4	232	210
12	19193	Patchaiammal	11.2	11.2	179	174
13	19195	Kumaravel	9.8	10.4	200	200
14	19536	Ilango	9.6	10.2	292	260
15	19619	Azhagu	13	13	174	180
16	19622	Perumal	10.4	10.6	127	130
17	19623	Parvathy	11	11	168	164
18	19627	Kannan	12	12	157	160
19	19535	Kumar	10.6	11	207	200
20	19625	Thangam	10.8	11	126	140

LABORATORY INVESTIGATION OF INPATIENTS

S.No	IP.No	Name	Hb gm%		Serum Cholesterol mg%	
			BT	AT	BT	AT
1	1051	Ramaya	8.5	8.6	138	120
2	1050	Iyyasamy	11.5	11.4	207	180
3	1256	Murugan	9.5	9.6	152	150
4	1283	Petchikumar	10.2	10.4	198	180
5	694	Subramanian	10.4	10.2	228	210
6	3348	Mani	8.5	8.6	130	130
7	1435	Muruga pandi	11	11.2	191	180
8	1411	Pon perumal	11.8	11.8	233	200
9	1256	Murugan	9	9.2	112	112
10	845	Mariadass	10.4	10.6	176	160
11	135	Sankar	10.4	10.6	96	100
12	69	Shanmugam	9.8	10	148	140
13	26	Shanmugaiah	10.5	10.4	160	160
14	1138	Rajalakshmi	9.8	10	159	140
15	500	Mariyal	9.6	9.8	142	142
16	3554	Shanmugam	10	10.2	230	210
17	777	Chermakani	9	9.2	192	180
18	900	Mary	10.2	10.2	228	210
19	3294	Lakshmanan	10.5	10.5	180	170
20	3348	Mani	10	10.2	270	250

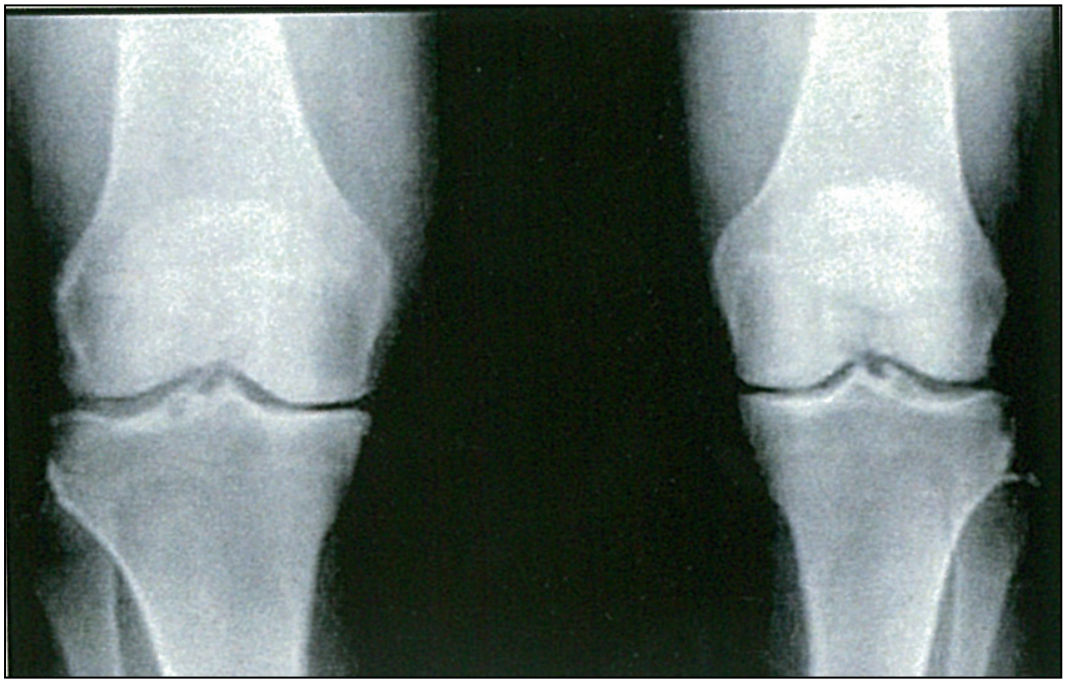
CASE SUMMARY OF OUT PATIENTS

S.NO	OP. NO	NAME	AGE	SEX	DOA	DOI	DOD	TREATED DAYS	RESULT
1	14176	Alagammal	48	F	11.02.16	1Year	22.03.16	40	Good
2	14178	Annammal	65	F	11.02.16	6 months	20.03.16	38	Good
3	14179	Anbalagan	56	M	11.02.16	2 years	24.03.16	42	Good
4	15681	Annammal	55	F	16.02.16	7 months	27.03.16	40	Good
5	15682	Poongothai	48	F	16.02.16	2 months	25.03.16	38	Good
6	17161	Chandrasekar	70	M	20.02.16	4 years	31.03.16	40	Fair
7	17185	Shanmugam	57	M	20.02.16	1 year	01.04.16	41	Good
8	18114	Boomadevi	53	F	23.02.16	6 months	02.04.16	39	Good
9	18116	Ponnammal	53	F	23.02.16	3 months	03.04.16	40	Good
10	18806	Isakkiammal	60	F	25.02.16	2 months	06.04.16	41	Fair
11	18919	Prakasi	60	F	25.02.16	8 months	07.04.16	42	Good
12	19193	Patchaiammal	65	F	26.02.16	6 months	05.04.16	38	Good
13	19195	Kumaravel	58	M	26.02.16	4 years	02.04.16	36	Good
14	19536	Ilango	48	M	27.02.16	9 months	07.04.16	40	Good
15	19619	Azhagu	49	F	27.02.16	1 year	09.04.16	42	Good
16	19622	Perumal	58	M	27.02.16	8 years	05.04.16	38	Fair
17	19623	Parvathy	55	F	27.02.16	6 months	07.04.16	40	Good
18	19627	Kannan	58	M	27.02.16	4 months	09.04.16	42	Good
19	19535	Kumar	65	M	27.02.16	3 years	07.04.16	40	Good
20	19625	Thangam	65	F	27.02.16	2 months	05.04.16	38	Good

CASE SUMMARY OF IN PATIENTS

S.NO	IP NO	NAME	AGE	SEX	DOA	DOI	DOD	TREATED DAYS	RESULT
1	1051	Ramaya	65	M	19.04.16	2 years	14.05.16	25	Good
2	1050	Iyyasamy	65	M	19.04.16	14 months	05.05.16	16	Fair
3	1256	Murugan	39	M	12.05.16	16 months	15.06.16	35	Good
4	1283	Petchikumar	38	M	17.05.16	6 months	18.06.16	33	Good
5	694	Subramanian	72	M	14.03.16	3 years	08.04.16	25	Good
6	3348	Mani	59	M	29.12.15	8 months	19.01.16	22	Good
7	1435	Muruga pandi	60	M	21.05.16	2 years	16.06.16	25	Good
8	1411	Pon perumal	49	M	29.04.16	6 years	21.05.16	23	Fair
9	1256	Murugan	63	M	12.05.16	18 months	10.06.16	29	Good
10	845	Mariadass	55	M	29.03.16	3 years	28.04.16	30	Good
11	135	Sankar	55	M	22.01.16	6 months	19.02.16	27	Good
12	69	Shanmugam	60	M	16.01.16	9 years	05.02.16	20	Good
13	26	Shanmugaiah	80	M	28.12.16	15 months	18.01.16	22	Good
14	1138	Rajalakshmi	60	F	27.03.16	3 years	14.04.16	19	Fair
15	500	Mariyal	63	F	25.02.16	1 year	20.03.16	24	Good
16	3554	Shanmugam	67	M	23.12.15	2 years	15.01.16	23	Good
17	777	Chermakani	37	F	23.03.16	18 months'	12.04.16	21	Good
18	900	Mary	55	F	02.04.16	6 years	27.04.16	25	Fair
19	3294	Lakshmanan	61	M	01.02.16	3 years	27.02.16	17	Good
20	3348	Mani	72	M	26.12.16	7 years	18.01.16	23	Good

X – RAY OF OUTPATIENTS

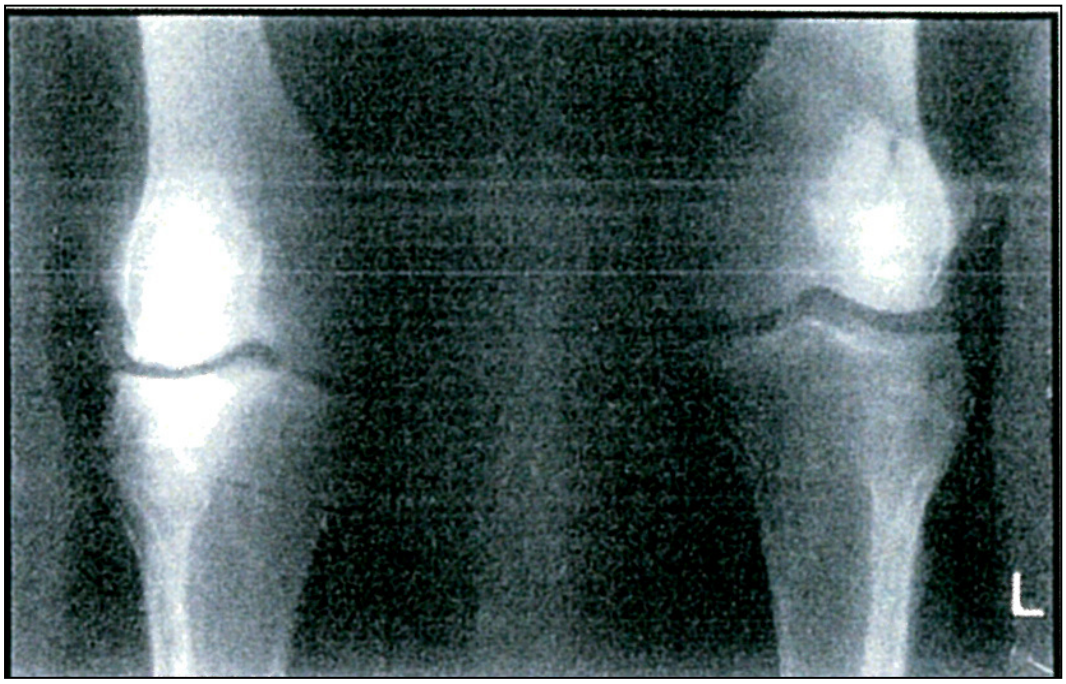


Op. No : 14176 Name : Alagammal 48 / F D.O.A: 11.02.2016

Op. No : 14179 Name : Anbalagan 56 / M D.O.A: 11.02.2016

X – RAY OF IN PATIENTS

Ip. No : 1051 Name : Ramaiah 65 / M D.O.A: 19.04.2016



Ip. No : 1050 Name : Ayyachamy 65 / M D.O.A: 19.04.2016